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THE GASTRIC ULCER PROBLEM *

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The treatment of gastric ulcer continues to be a source of controversy between the proponents of the time-honored methods of medical management and the advocates of prompt surgical exploration of all ulcerating lesions of the stomach. Strode [1] has recently stimulated our interest in this controversy by his aggressive stand in favor of prompt surgical intervention without preliminary medical trial.

At Vanderbilt University Hospital during the last fifteen or more years the treatment of gastric ulcer has theoretically followed the generally accepted pattern of a trial of medical management consisting of diet, antacids and antispasmodics for a period of six to eight weeks with surgical intervention if the ulcer fails to heal as evidenced by relief of symptoms and findings on repeated roentgen examinations. In order to assess the efficacy of this plan a study has been made of 186 patients with gastric ulcer seen at Vanderbilt University Hospital during the fifteen year period from 1940 through 1954. The statistical data from this study has been arranged in three categories for purposes of comparison: (1) the malignant ulcer group, (2) the benign gastric ulcers treated surgically and (3) the medically managed gastric ulcers.

MALIGNANT ULCERS

Twenty-two patients, or 11.8 per cent of the group, proved to have malignant ulcers. Table 1 shows that there were 21 males and one colored female. Ninety-five per cent of the cases came to operation and were proven histologically while one case was treated entirely by medical management and carcinoma was discovered at autopsy. This was a case of a 72 year old white male whose ulcer was demonstrated on the lesser curvature of the stomach 3 cm. proximal to the pylorus by G.I. series. Within six days of medical regimen the ulcer appeared to be healed by x-ray examination. The patient was entirely asymptomatic after two weeks of treatment and remained so for the next eight months. Ten months after his ulcer was diagnosed he died with carcinoma of the stomach.

TABLE 1
ANALYSIS OF 22 MALIGNANT ULCERS

Malignant ulcers in series—11.8%		
Operated	21	(95%)
Treated medically as benign	1	
Males	21	
White	17	
Negro	4	
Females	1	

The age incidence of malignant ulcers ranged from 35 years to 72 years. The

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majority were between 50 and 70 years old, but 6 patients or 27 per cent were under 50 years of age. There was only one female in this group of malignant ulcers although there was a total of 42 women in the entire series.

Table 2 shows the types of operation

TABLE 2
ANALYSIS OF 21 OPERATED MALIGNANT ULCERS: TYPE OF OPERATION

	No. Cases
Total gastrectomy	4
Subtotal gastrectomy	11
Exploratory laparotomy and biopsy	5
Gastroenterostomy	1
Resectable at time of operation—71%	

used for malignant ulcers. Almost one-third of the cases were not resectable at the time of operation.

In Table 3 is seen the length of medical

TABLE 3
ANALYSIS OF 21 OPERATED MALIGNANT ULCERS: LENGTH OF MEDICAL TREATMENT PRIOR TO OPERATION

Duration	No. Cases	
1 month or less	7	38%
1 to 2 months	1	
2 to 4 months	3	
4 to 6 months	0	
6 to 12 months	5	
1 to 2 years	3	
2 to 5 years	1	
19 years	1	
	21	

management preceding operation. Of interest is the fact that only 38 per cent of these patients were operated upon within two months of the time that a gastric ulcer was definitely diagnosed. One patient was followed repeatedly with roentgenologic examinations for one year before a prepyloric carcinoma was found at operation. In the three and one-half years since operation he has done very well and shows no evidence of recurrence. The lesion was a very early adenocarcinoma arising in an old chronic peptic ulcer.

The patient who was followed for nineteen years had a lesser curvature ulcer treated by diet with relief of symptoms for eighteen years. After development of recurrent symptoms, he was again referred to Vanderbilt University Hospital. A biopsy of the recurrent ulcer by gastroscopy revealed carcinoma. Total gastrectomy was performed, but the patient died three weeks after operation from a disruption of the esophagojejunal anastomosis. There was no evidence of metastatic spread at autopsy. This is the only operative death in the malignant ulcer series.

All 5 patients who were operated upon twelve months or more after diagnosis of gastric ulcer had been followed clinically and radiologically with medical therapy for their ulcers. Two were finally operated upon because of radiological recurrence, 2 because of increasing pain, and 1 because of symptoms of obstruction.

In Table 4 are mortality statistics in

TABLE 4
ANALYSIS OF 21 OPERATED MALIGNANT ULCERS: MORTALITY STATISTICS

Total operative mortality	1 *

* Disruption of anastomotic site after total gastrectomy.

the malignant ulcer group. As mentioned above there was only one operative death.

Table 5 shows the survival period in the

TABLE 5
ANALYSIS OF SURVIVAL PERIOD IN 15 RESECTABLE CASES

	No. Cases	%
2 to 5 years	3	20
More than 5 years*	4	26.6
Dead in 1 year or less	5	
Dead in 1 to 2 years	3	

* 1 died of metastases 8 years postoperatively; rest are alive 6 years, 7 years and 11 years after operation.

15 resectable cases. One-fourth survived more than five years. One patient is living and well eleven years after resection, one seven years, and one six years. One patient was reported to have had a benign ulcer on microscopic examination but died eight years later with liver metastases and carcinoma of the stomach. At the time of autopsy a review of the original sections showed the surgical pathological diagnosis to have been in error as carcinoma was indeed present.

BENIGN GASTRIC ULCERS TREATED SURGICALLY

There were 164 benign ulcers. These are subdivided into 92 cases treated medically and proved benign only by the patient's clinical course and roentgen findings, and 72 cases treated by definitive

gastric surgery. Seventy of these 72 cases had excision of the ulcer and pathological examination to prove the lesion benign. One patient had a gastrojejunostomy without resection. His operation was done in 1943 because of obstructive symptoms. Twelve years later he is alive and well. Another patient had a benign ulcer located high on the lesser curvature which was treated by gastroenterostomy and vagotomy.

Table 6 shows the sex ratio breakdown in this group. There were 54 males as compared to 18 females.

TABLE 6
ANALYSIS OF 72 BENIGN OPERATED GASTRIC ULCERS

Males		54
White	48	
Negro	6	
Females		18
White	17	
Negro	1	

The age range in the group is shown in Table 7. The majority of patients were in the fifth through the seventh decades.

TABLE 7
ANALYSIS OF 72 BENIGN OPERATED GASTRIC ULCERS: AGE AND SEX

Age Years	No. Cases Male	Female
30 to 39	5	1
40 to 49	14	5
50 to 59	17	5
60 to 69	15	6
70 to 79	3	1
	54	18

Table 8 lists the indications for operation. Almost one-third of the patients were operated on because of a strong suspicion of malignancy. One-third of the cases were operated upon because of intractability, hemorrhage, or obstruction.

TABLE 8
ANALYSIS OF 72 BENIGN OPERATED GASTRIC ULCERS: INDICATIONS FOR OPERATION

	No. Cases	
Diagnosis of carcinoma	8	
Diagnosis of probable carcinoma	15	(31.9%)
Recurrence	4	(5.5%)
Obstruction	3	
Hemorrhage	6	(33.3%)
Intractability	15	
Failure to heal	15	(20.8%)
Perforation	2	
Resection planned D. U. because intractability—found G.U. also	4	

Table 9 lists the types of operations. The Billroth II anastomosis was done routinely on all staff patients during the first ten years of this study.

TABLE 9
ANALYSIS OF TYPES OF OPERATION FOR 72 BENIGN ULCERS

	No. Cases
Billroth II	38
Hofmeister	18
Polya	9
Billroth I	2
Vagotomy and Billroth I	1
Vagotomy and Polya	1
Billroth II and vagotomy	1
Gastrojejunostomy	1
Gastrojejunostomy and vagotomy	1

The mortality figures are shown in Table 10. Two of the three deaths are considered to be the direct result of the operation. The operative mortality rate is therefore 2.8 per cent. One other patient died while in the hospital of cardiac failure.

TABLE 10
ANALYSIS OF 72 DEFINITIVE OPERATIONS: MORTALITY STATISTICS

Mortality	Cause of Death
1	Peritonitis, leak gastrojejunostomy suture line, 12 days postoperatively, 69 year old white male.
1	Multiple lung abcesses, generalized peritonitis, 25 days postoperatively, 52 year old female.
1	Cardiac failure, 24 days postoperatively.
3 = 4.2%—Hospital deaths Operative mortality = 2.8%	

In Table 11 may be seen the length of

TABLE 11
ANALYSIS OF 72 OPERATED BENIGN ULCERS: LENGTH OF MEDICAL TREATMENT PRIOR TO OPERATION

Duration	No. Cases	
1 month or less	52	
1 to 2 months	5	79.2%
2 to 4 months	1	
4 to 6 months	2	
6 to 12 months	3	
1 to 2 years	5	
2 to 4 years	3	
4 to 6 years	1	
	72	

medical management prior to operation. Fifty-seven patients or 79.2 per cent were operated upon within two months or less after the diagnosis of gastric ulcer was made.

Table 12 analyzes the follow-up of the surgically treated benign ulcer cases and classifies the results in relation to length of follow-up.

Two proven cases of recurrent ulceration have resulted. One of these had marginal recurrence within four months of operation. This patient did fairly well on medical regimen for a year but recently his persistent marginal ulcer was successfully resected. The other patient developed a gastrojejunocolic fistula two years after partial gastrectomy. Following surgical correction of this he has done very well for the past seven years. The third poor result was in a patient who had hematemesis two years after operation at which time a vagotomy was done for suspected marginal ulcer. Six years later hematemesis again occurred and a higher gastric resection was performed. No recurrent ulceration was found, and pathological examination reported chronic inflammation of the stomach. His subsequent course has been satisfactory.

Three patients have died of heart disease from sixteen months to six years after their operation. This can have no relation to their classification of postoperative status.

MEDICALLY MANAGED GASTRIC ULCERS

The group of medically managed gastric ulcers totals 92 cases. One patient who was treated medically and proved subsequently to have carcinoma at death has been included in Table 1 and is not included in these 92 cases.

Table 13 shows the sex ratio in this group. There was a ratio of 3:1 for males to females.

TABLE 13
ANALYSIS OF 92 BENIGN GASTRIC ULCERS WITH MEDICAL MANAGEMENT

Males		69
White	64	
Negro	5	
Females		23
White	17	
Negro	6	

In Table 14 is seen the age range for this group. There is a wide range but the majority fall in the fourth through the seventh decades.

TABLE 14
ANALYSIS OF 92 BENIGN GASTRIC ULCERS WITH MEDICAL MANAGEMENT

Ages - Years	Male	Female
10 - 19	1	0
20 - 29	5	0
30 - 39	8	5
40 - 49	7	5
50 - 59	19	2
60 - 69	23	4
70 - 79	5	6
80 - 89	1	1
	69	23

Table 15 analyzes the follow-up in this group. Fourteen patients have had no follow-up and 36 have been followed less than two years. Ten of these have died of known causes: two of heart disease, and one each of trauma, hemorrhage from gastric ulcer, pulmonary edema, bronchogenic carcinoma, ruptured appendix, Addison's disease, amebic abscess of the liver, and retroperitoneal tumor.

TABLE 12
ANALYSIS OF 72 OPERATED BENIGN ULCERS: FOLLOW-UP OF CASES

	No. Cases	Results		
		Poor	Fair	Good
Died in hospital	3	—	—	—
No follow-up	3	—	—	—
Less than 1 year postoperatively	16	1		15
1 to 2 years postoperatively	13			13
2 to 3 years postoperatively	8			8
3 to 4 years postoperatively	5			5
4 to 5 years postoperatively	6			6
5 to 10 years postoperatively	14	2	3	9
10 to 12 years postoperatively	4			4

TABLE 15
ANALYSIS OF 92 CASES DIAGNOSED AS BENIGN AND TREATED MEDICALLY

	No. Cases	Deaths Known	Deaths Unknown
No follow-up	14		
Less than 2 yrs.	36	10	
2 to 5 yrs.	19	3	
5 to 10 yrs.	16	2	1*
10 yrs. or more	7	1	
Total	92	16	1

* Died age 62 yrs.

Of the patients followed two to five years three died of known causes: two of heart disease and one of glomerulonephritis. In the group followed five to ten years, three have died: one of heart disease, one of carcinoma of the breast, and one at age 62 years of an unknown cause. One patient died of heart disease in the group of seven that was followed over ten years.

The incidence of recurrence in each group with a follow-up period is shown in Table 16. Since 5 of the 15 recurrences were not demonstrated by x-ray, the recurrence rate is shown in those cases without associated duodenal ulcer so that the recurrence cannot be attributed to activity in an associated duodenal ulcer. It is of interest to note that the percentage of recurrence rises as the time of follow-up lengthens.

TABLE 17
ANALYSIS OF 15 PATIENTS WITH RECURRENCE WHO DID NOT HAVE SURGERY

	No. Cases
Did not return at time of recurrence	2
Refused operation	6
Had single recurrence with rapid recovery	7
	15

Table 17 gives an analysis of the 15 patients who had recurrences. Recurrence is generally considered as an indication for operation, and this table shows findings in the group not operated upon.

COMPARISON BETWEEN BENIGN AND MALIGNANT GASTRIC ULCERS

Figures 1 and 2 show the location of malignant and benign ulcers in this study. Figure 3 shows the percentage of malignant ulcers, obtained by dividing the number

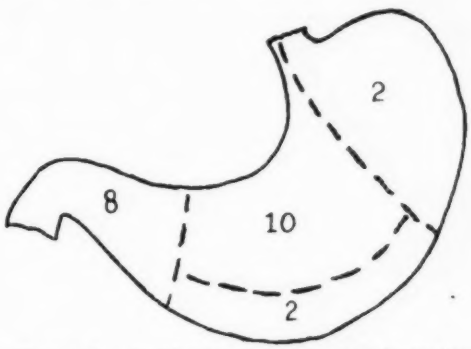

Figure 1. Incidence of malignancy in various locations. Malignant ulcers 22.

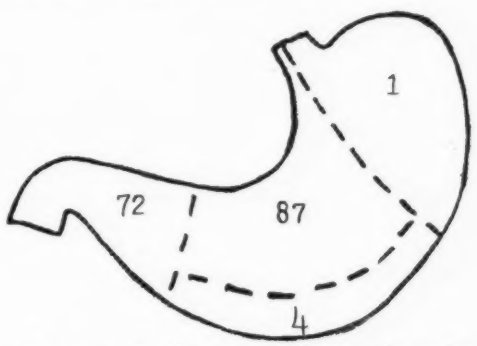

Figure 2. Incidence of malignancy in various locations. Benign ulcers 164.

TABLE 16
ANALYSIS OF 92 CASES DIAGNOSED BENIGN AND TREATED MEDICALLY: INCIDENCE OF RECURRENCE

Length Follow-up	Total No. Cases	No. Dx by X-ray	No. Dx By Sympt. & X-ray	% Recurrence	Without Duodenal Ulcer: Total Cases	Without Duodenal Ulcer: Recurrence No.	Without Duodenal Ulcer: Recurrence %
None	14	—	—	—	—	—	—
Less than 2 years	36	—	2	5.5	20	2	7.2
2 to 5 yrs.	19	4	5	26.3	15	5	33.3
5 to 10 yrs.	16	4	5	31.3	12	3	25.0
10 yrs. or more	7	2	3	42.9	7	3	42.9
Total No. with follow-up	78	10	15	19.2	54	13	24.1

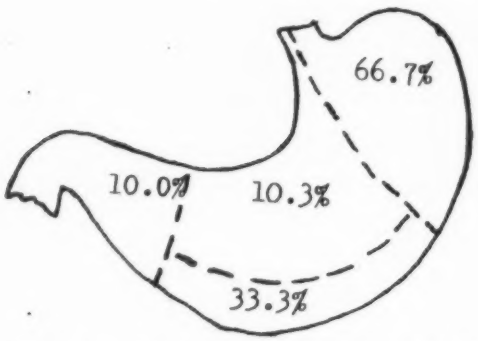

Figure 3. Incidence of malignancy in various locations.

of malignant ulcers by the total number for each location.

In Table 18 a comparison of the findings of gastric analysis is made in patients with benign and malignant ulcers. Anacidity is not incompatible with benign ulcer but is more than three times as frequent in patients with malignant ulcer. No patient with malignant ulcer had free acid over 51 degrees while 5.5 per cent of the benign cases were over this figure. About half of the patients in each group had gastric analysis performed. The results shown are those after histamine or alcohol stimulation.

TABLE 18
COMPARISON OF GASTRIC ACIDITY IN 22 MALIGNANT ULCERS AND 164 BENIGN ULCERS

	Malignant		Benign	
Acidity	No. Cases	Per Cent	No. Cases	Per Cent
Anacidity	6	27.2	12	7.3
Less than 25	1	4.5	38	23.2
26 to 50	5	22.7	21	12.8
51 and over			9	5.5
Not recorded	10	45.4	84	51.2

TABLE 19
COMPARISON OF WEIGHT LOSS IN 22 MALIGNANT ULCERS AND 164 BENIGN ULCERS

	Malignant		Benign	
	No. Cases	Per Cent	No. Cases	Per Cent
10 lbs. or less	9	40.9	116	70.7
11 to 20 lbs.	3	13.6	6	3.6
21 to 30 lbs.	1	4.5	2	1.2
31 or more	4	18.1	6	3.6
Wt. loss but amt. unknown	4	18.1	34	20.7
Gained 10 lbs.	1	4.5		

Comparison of weight loss is shown in the next table. The malignant ulcer group had a much higher percentage of weight loss than did the benign group.

Complications of hemorrhage, obstruction, and perforation are shown in Table 20. There were no perforations in the malignant ulcer group.

TABLE 20
COMPARISON OF COMPLICATIONS IN 22 MALIGNANT ULCERS AND 164 BENIGN ULCERS

	Malignant		Benign	
	No. Cases	Per Cent	No. Cases	Per Cent
Hemorrhage	7	31.8	48	29.2
Obstruction	2	9.1	4	2.4
Perforation	0	0	13	7.9

Table 21 gives a detailed analysis of the benign perforated gastric ulcers. There were no operative deaths. However, only two of the patients with adequate follow-up had a satisfactory course as regards subsequent ulcer behavior and one of these died from heart disease eight months after closure of his perforation.

TABLE 21
ANALYSIS OF PATIENTS WITH PERFORATED GASTRIC ULCERS

Operation	No. Cases	Op. Death
Closure perforation	13	0
Results		
Satisfactory course	2*	
Unsatisfactory course	8	
Subtotal resection		3
Persistent pain		3
Died of amebic abscess 3½ mo. P.O.		1
Died of retroperitoneal tumor 1 mo. P.O.		1
Lost to follow up	3**	

* 1 doing well 9 yrs. after closure, 1 died 8 mo. postop. of heart disease.
** 1 doing well at 6 months.

As can be seen in Table 22, duodenal ulcer occurred in 9.1 per cent of the malignant ulcer cases and in 15.8 per cent of the benign ulcer cases. This shows the fallacy of the often quoted maxim

TABLE 22
ASSOCIATED FINDINGS IN 22 MALIGNANT ULCERS AND 164 BENIGN ULCERS

	Malignant		Benign	
	No. Cases	Per Cent	No. Cases	Per Cent
Duodenal ulcer	2	9.1	26	15.8

that malignant gastric ulcer is not seen with an associated duodenal ulcer.

Table 23 shows the comparison of presenting symptoms in each group. Epigastric discomfort was not tabulated for the benign group as a separate entity but is included under pain. This table emphasizes the inability of differentiation between malignant and benign ulcers by symptomatology.

TABLE 23
COMPARISON OF PRESENTING SYMPTOMS IN 22 MALIGNANT ULCERS AND 164 BENIGN ULCERS

	Malignant		Benign	
	No. Cases	Per Cent	No. Cases	Per Cent
Pain	20	90.9	149	90.9
Vomiting	10	45.4	76	46.3
Dysphagia	2	9.1		
Epigastric discomfort	6	27.2		
Bloating	1	4.5	2	1.2
Weakness			1	0.6
Ascites			1	0.6
Anemia			1	0.6
Nausea			1	0.6

The results of gastroscopy are shown in Table 24. Lesions were visualized in 60 per cent of the cases examined with an accuracy rate of 85.7 per cent. However, it must be noted that in 40 per cent of the patients who were gastroscoped no lesion was seen even though a gastric ulcer was present. In no case was an ulcer demonstrated by gastroscopy which was not also visualized by radiologic examination.

TABLE 24
PATIENTS WHO HAD GASTROSCOPY PERFORMED (35 CASES)

	Cases	Per Cent
Examination unsatisfactory	14*	40
Lesions visualized	21	60
DX benign and confirmed	15**	71.4
DX benign and proved malignant	1	4.7
DX ? malignant and proved benign	2	9.5
DX malignant and proved malignant	3	14.3
85.7% accurate. 14.2% inaccurate.		

* Two patients had two gastroscopies with failure to visualize ulcer.

** In one case ulcer was not seen on initial examination.

COMMENT

In this group of 186 patients with gastric ulcer the incidence of malignancy was 11.8 per cent. This incidence is similar to that encountered in other larger series. Smith and Jordan [2] in a review of 600 gastric ulcers at the Lahey Clinic found that 9.8 per cent were malignant. Cain, Jordan, Comfort and Gray [3] at the Mayo Clinic reported a 10.4 per cent incidence of carcinoma in 414 gastric ulcers, while Allen [4] found that 14 per cent of patients treated as having benign gastric ulcers at the Massachusetts General Hospital proved to have malignant lesions.

A study such as this demonstrates that the crux of the gastric ulcer problem is the impossibility of definitely differentiating benign from malignant lesions by any other means than microscopic examination of the excised ulcer. It further emphasizes the inadequacy of the generally accepted plan of medical management of gastric ulcer as it has been practically applied in this university hospital clinic in the last fifteen years. Only 26 per cent of the medically managed group of patients with gastric ulcer could be adequately followed by the desired program of rigid medical control and repeated roentgen examinations. A sense of complacency as regards gastric ulcer in relation to its malignant potential has pervaded the medically managed series of this study. The well-known fact that a small ulcerated gastric cancer may heal on a medical regimen has been reconfirmed by this survey.

The study has also shown that a high incidence of recurrence (24 per cent) with attendant hazardous complications has occurred in the medically managed gastric ulcers and that this incidence increases proportionally with the length of follow-up. In contrast, the incidence of recurrence after surgical treatment of benign gastric ulcer has been quite low (2.6 per cent) and the long range results have been generally good.

Consideration of the above findings with specific emphasis on the clinician's inability to differentiate benign from cancerous lesions in a disease which has an incidence of malignancy of 10 to 14 per cent leads us to the conclusion that the

generally accepted plan of medical management for gastric ulcer should be abandoned and that patients with ulcerating lesions of the stomach should have prompt surgical intervention without a preliminary trial of medical therapy.

SUMMARY

The following conclusions have been drawn from this statistical study of 186 patients with gastric ulcer:

1. The gastric ulcers in this series proved to be malignant lesions in 11.8 per cent.

2. Despite a clinic policy during the years of this study of strict medical regimen with frequent x-ray examinations to determine healing of the ulcer within the first six weeks after diagnosis, only 26 per cent of the medically managed group could be adequately followed.

3. It is not possible by present diagnostic methods to determine whether a gastric ulcer is malignant or benign until it is surgically removed and studied microscopically.

4. The incidence of recurrence and unfavorable results increases in direct proportion to the length of follow-up in the medically managed gastric ulcer group.

5. The long range results of partial gastric resection for benign gastric ulcer are generally good.

6. Therefore we conclude that patients with ulcerating lesions of the stomach should have surgical intervention as soon as the diagnosis is made without a preliminary period of medical management.

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SERUM TRANSAMINASE AS AN AID IN THE DIAGNOSIS OF MYOCARDIAL INFARCTION *

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NEW ORLEANS

The diagnosis of myocardial infarction by electrocardiographic criteria may often present many difficulties. Conduction defects such as left bundle branch block and Wolfe - Parkinson - White syndrome make the diagnosis of a recent myocardial infarction by the electrocardiogram extremely difficult. A similar problem can exist when multiple previous infarctions make significant alterations in the electrocardiogram undetectable. Because of the above-cited situations, many of the alterations in the blood of patients who have experienced a recent myocardial infarction have been studied by several investigators in the past. Some of these, such as fever, leukocystosis, an elevated erythrocyte sedimentation rate and plasma fibrinogen levels, the presence of C-reactive protein as well as alterations in blood coagulability, serum lipids, globulins, copper and iron are all nonspecific indications of an inflammatory process. They vary in their sensitiveness, and as a result of their nonspecificity all may be false indicators of myocardial necrosis if necrosis exists elsewhere in the body.[1]

MECHANISM OF SGO-T ACTIVITY

Recently, LaDue and co-workers have studied the properties of serum transaminase and have shown this enzyme to be elevated in the serum following an acute myocardial infarction.[2] The study of this tissue enzyme activity of the blood following a recent infarction may be a more direct approach of muscle necrosis. Also,

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this may prove to be a simple chemical test which will be of diagnostic help in recent infarction.

Glumatic oxalacetic transaminase is a specific tissue enzyme concerned with the transfer of alpha-amino nitrogen of aspartic acid to alpha-keto glutaric acid, resulting in the synthesis of a new amino-acid, glutamic acid and a new alpha-keto acid, oxalacetic acid.[3] SGO-T activity has been demonstrated in all human and animal sera tested. Heart muscle, skeletal muscle, brain, liver and kidney contain SGO-T in decreasing concentrations, although it is distributed widely in animal tissues. Injury to heart muscle, skeletal muscle, liver and kidney will cause increased activity of SGO-T in the serum. Serum transaminase activity has been found to be normal in infectious, degenerative, neoplastic, allergic, reactive or congenital disease states, unless damage to liver, heart or skeletal muscle is present.[4]

SGO-T activity in the blood increases 2 to 20 times normal after acute myocardial infarction. Experimental infarctions in dogs have shown that this rise in the blood is associated with a corresponding decrease in the transaminase concentration of the infarcted area.[5] Again, with myocardial infarction produced experimentally in dogs, the increase in activity occurs within six hours following the production of an infarction, and both the degree and duration of the enzyme abnormality appear to be proportional to the size of the infarction. Myocardial ischemia of forty-five minutes' duration did not influence the serum level of glutamic oxalacetic transaminase. This suggests that the mechanism of elevation of transaminase activity is simply one of release of the enzyme into the bloodstream following death or increase in cellular membrane permeability. The routes of excretion and degradation of the enzyme are not yet known.[6]

Serum transaminase activity in the blood may be increased 20 to 100 times normal in hepatitis due to toxic or infectious agents. SGO-T activity during the acute phase of hepatitis, whatever its cause, actually permits differentiation of jaundice due to this cause from that due to cirrhosis, extrahepatic obstruction of the biliary ducts, liver metastasis and hemolysis.[7]

STUDY OF 134 PATIENTS

The present study is a continuation of a previous study,[8] and was undertaken to evaluate the determination of serum transaminase activity as an ancillary procedure in the clinical diagnosis of myocardial infarction. One hundred and thirty-four patients were studied. The spectrophotometric method of determination was used.[3] Approximately 75 per cent of the patients in this series represented admissions to the Louisiana State University Medical Service at Charity Hospital at New Orleans. The remaining 25 per cent were patients of physicians in private practice. There were 35 patients with established diagnoses other than recent myocardial infarction or liver disease, who served as a control group. The range of SGO-T activity in this control group varied from 6 to 40 units, the mean being 16.2 units. Values exceeding 50 units were considered to be elevated, this being the lowest level observed in the group with clinical and electrocardiographic manifestations of myocardial infarction. The remaining patients presented with chest pain of a nature that the diagnosis of a recent infarction was suspected. Of these patients, 47 had clinical and electrocardiographic evidence of acute myocardial infarction. Forty-five of 47 patients in this particular group had SGO-T levels above 50 units, varying from 50 to 266 units. The levels became increased as early as four hours and as late as three days following the onset of chest pain. Thirteen of these patients expired, but postmortem examination was obtained in only 6 of these. All 6 exhibited gross and microscopic evidence of recent myocardial infarction by postmortem examination.

Of the 2 patients with clinical and electrocardiographic evidence of acute myocardial infarction whose transaminase levels were in the normal range, 1 had re-

peated specimens drawn, the maximum level being 40 units. The other patient had evidence of a recent massive transmural infarction, both electrocardiographically and by postmortem examination. The patient expired seven hours after the onset of pain, and the specimen had been drawn six hours after the onset of pain.

Three patients with electrocardiographic changes indicating the presence of a conduction defect, namely left bundle branch block, but without definite electrocardiographic evidence of the change of a recent myocardial infarction, were noted to have SGO-T levels above 50 units. All 3 patients had clinical manifestations of myocardial infarction.

Of the remaining, 49 had suspected myocardial infarctions not verified by electrocardiogram. All had serum transaminase levels under 50 units, varying from 10 to 48 units. Electrocardiographic patterns in this group varied from normal electrocardiographs to patterns of ischemia.

These observations suggest a correlation between serum transaminase activity and electrocardiographic changes in recent myocardial infarction and indicate the usefulness of this laboratory procedure as an aid to the diagnosis of myocardial infarction.

CONCLUSIONS

1. We wish to point out that this laboratory test definitely does not attempt to replace clinical judgment or the electrocardiogram in the diagnosis of a recent myocardial infarction. It should be integrated into the general picture.

2. This laboratory test may be the advance guard in the development of a highly sensitive and specific blood chemical test for the detection of a recent myocardial infarction.

3. The greatest value of this test will be in those cases with conduction defects where the electrocardiogram may be of little help in the diagnosis of an acute infarction. Further study with this group is indicated, supplemented by postmortem studies.

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SHIGELLOSIS *

A REVIEW OF SHIGELLOSIS CASES ADMITTED TO THE PEDIATRIC SERVICES OF CHARITY HOSPITAL AT NEW ORLEANS DURING A ONE-YEAR PERIOD, 1955

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Shigellosis has been a common cause of disease and death since early times. Better standards of living, improved sanitation, and modern therapeutic measures have drastically reduced morbidity and mortality due to this disease. Because of these changes many physicians may not be aware of the problems shigellosis still presents today, and for this reason a review of our experiences with this disease during 1955 was deemed worthwhile.

This report is primarily concerned with patients admitted to the pediatric services at Charity Hospital of Louisiana in New Orleans between January 1, 1955, and December 31, 1955. This group represents

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only 62 of 222 hospitalized or clinic patients of all ages from whom Shigella organisms were isolated. These 62 patients represent 11 per cent of the 575 patients admitted to the pediatric services with the diagnosis of diarrhea.

The cultures were obtained by rectal swab and were studied by the usual microbiological techniques. The rectal swab was streaked on a quadrant of an SS plate and was further streaked by bacteriological loop through remaining quadrants. After twelve to eighteen hours' incubation at 37° C., at least three colonies which neither fermented lactose nor produced H_2S were inoculated into Kligler's iron agar and urea broth (Difco). After twenty-four hours' further incubation those colonies showing fermentation reaction typical of Shigellae in Kligler's medium and failing to hydrolize urea were tested for agglutination by a slide technique using commercial type-specific Shigellae antisera (Lederle). In practically all instances only Group B and D sera were used. In all cases identfication was confirmed by biochemical activity of the strains, including carbohydrate fermentations, indol production and citrate fermentation, and motility in a semi-solid medium.

CLINICAL MANIFESTATIONS

Review of the major clinical manifestations revealed diarrhea, fever, and blood in the stools to be the most common signs and symptoms. (Table 1) Every case except one had evidence of diarrheal stools. The stools were described as watery in consistency and yellow or green in color. Macroscopic blood in the stool was present in 50 per cent of the cases, either by history or observation of the stools during hospitalization. Ninety percent of the patients demonstrated a temperature of 100° F. or over sometime during their illness.

TABLE 1
MAJOR CLINICAL MANIFESTATIONS OF 62 PATIENTS WITH SHIGELLOSIS

Diarrhea	61
Temperature over 100°	56
Blood in stools	31
Convulsions	19
Delirium	6

It soon became evident that these cases could easily be divided into two rather well-defined clinical pictures. (Table 2) Those children under 3 years of age usually followed a pattern of a gradual or insidious onset with diarrhea for two to seven days or longer before they were admitted to the hospital. An intermittent fever existed for several days before admission. There were few other distinguishing features. Many of these patients were not brought to the hospital for treatment until blood was noticed in the stools.

TABLE 2
DIFFERENCES IN CLINICAL MANIFESTATIONS BY AGE GROUPING IN 62 PATIENTS WITH SHIGELLOSIS

	Under 3 yrs.	3 yrs. or over
Total	36	26
Diarrhea	35	26
Temp. over 100°	31	25
Abrupt onset*	12	21
Blood in stools	17	14
Convulsions	6	13
Delirium	0	6

*Abrupt onset: Onset of symptoms 24 hours or less prior to admission to hospital.

The children 3 years of age or older presented a more acute illness, with sudden onset, high fever, and toxicity. Convulsions occurred in 50 per cent of these children. Many of these patients did not develop diarrhea until after they arrived on the ward.

No difference in symptomatology was found to relate to any other factor including the comparison of cases of *Shigella flexneri* and cases of *Shigella sonnei.*

THERAPY

The therapy used in these cases consisted of parenteral fluid, specific antibiotic or chemotherapeutic agents, and sedation. Thirty-eight of the 62 cases warranted the use of intravenous fluids. Sedation was limited to patients with CNS manifestations. All patients were treated with one or more specific agents.

Sulfadiazine or a triple-sulfonamide mixture was used in the majority of cases. (Table 3) A combination of sulfonamides and chloramphenicol was the second most common regimen. All but one case responded well clinically on initial therapy.

TABLE 3
ANTIBIOTIC OR CHEMOTHERAPEUTIC AGENTS USED IN 62 PATIENTS WITH SHIGELLOSIS

One of sulfa drugs	44
Sulfa + chloramphenicol	11
Chloramphenicol	4
Sulfa + tetracycline	1
Sulfa-chloro-tetra	1
Sulfa-chloro-oxytetracycline	1

In cases where follow-up cultures were obtained the results mirrored the clinical response.

The usual course of these patients was early clinical improvement with complete absence of signs and symptoms by the fourth day of therapy. Only 8 patients required longer convalescence, and they had shown marked improvement by the fourth day.

One child who did not follow the usual clinical course on sulfonamide therapy was found to harbor a strain of *Shigella flexneri* which was resistant to the various sulfonamides, according to *in vitro* sensitivity tests. This strain was sensitive to chloramphenicol and tetracycline. After two days of therapy with chloramphenicol this child became asymptomatic, but seven additional days of therapy did not eradicate the organism from the stool. The child was placed on tetracycline for five days, during which time stool cultures became negative for Shigella.

FAMILIAL INFECTION

Stool cultures were obtained from members of the families of 18 patients. (Table 4) It was not possible to obtain a culture from every member of each family. Only one culture was obtained from each person investigated. In 15 families at least one person other than the index case was found to harbor Shigella organisms. The same type of organism found in the index case of each family was recovered from 24 of the 43 family members cultured.

TABLE 4
STUDY OF FAMILIES OF PATIENTS WITH SHIGELLOSIS

Families cultured	18
Families with at least one member with positive culture	15
Total number of family members cultured	43
Family members with positive culture	24

None of above figures includes the index cases.

DISCUSSION

From this review, 1 out of every 10 children with diarrhea admitted to this hospital was found to have shigellosis. This probably is not the true incidence of Shigella infections in this community. Only patients with severe or unusual symptoms were picked from our large out-patient service for admission. The incidence of shigellosis found also may have been influenced by diligence in the search for the organism. Since this was not a research project, repeated anal swabs were not obtained on many patients found to have a negative culture on the first attempt. Both of these factors may have influenced the results. The 11 per cent of the diarrhea patients with shigellosis found here is not comparable to 44 per cent reported by Hardy and Watt[1] (New Mexico, 1936-38; Georgia, 1939-40; New York, 1939-40 and 1942) or the 49 per cent reported by Cooper *et al.*[2] (Cincinnati, Ohio, 1938). It can be said, however, that shigellosis is a rather common problem in children at Charity Hospital of Louisiana.

The clinical manifestations listed here are the same as given elsewhere and reflect the point made by Hardy and Watt that the signs and symptoms presented by patients with shigellosis are not distinctive enough to differentiate clinically the cases from other types of diarrhea.[3]

CNS disturbance such as convulsions or delirium occurred in 40 per cent of the patients. This is a much higher incidence than is usually encountered in patients hospitalized with other types of diarrhea, and agrees with observations reported by Donald *et al.*[4] The predominance of these symptoms in the other age group appears to be significant.

It cannot be assumed that the good clinical response of patients was due to the antibiotic or chemotherapeutic agent used. This point could not be adequately assessed as a control group was not used. It is interesting that 6 patients who did not receive specific therapy until the third to fifth day of hospitalization showed clinical

improvement before such agents were administered. The degree of improvement was comparable to that of patients receiving specific therapy. However, Hardy and associates found that the clinical response of patients receiving sulfonamides and nonspecific therapy was definitely slower and more irregular than the response of patients receiving antibiotics effective against the particular strains of organisms.[5] Further questions concerning the actual role of sulfonamides may be obtained from the study of Tateno. He demonstrated that while 13 of 14 strains of *Shigella flexneri* isolated in the United States before 1952 were sensitive to sulfadiazine, only 2 of 8 strains isolated in 1953 were sensitive.[6] All of the strains isolated in the United States in his study were sensitive to streptomycin, chloramphenicol, oxytetracycline, chlortetracycline, and tetracycline by *in vitro* studies.

Hardy and associates, in reporting on a study in Korea, concluded that aureomycin, chloramphenicol, and terramycin are all effective in shigellosis.[5] Tetracycline was not available at that time. Sulfonamides were of little use in their study as most of the strains of Shigella encountered were resistant.

One of the most interesting, and possibly least appreciated, aspects of the shigellosis problem is the relationship of familial infection to the given case. In order to gain more understanding of this aspect of the disease, cultures were obtained from all available members of the families of 18 cases of shigellosis. Shigella organisms were recovered from a family member other than the index case in over 80 per cent of the families. Over 50 per cent of family members cultured harbored Shigella organisms of the same type found in the index case. Most of these familial contacts were asymptomatic at the time of the culture. Historical data were not adequate to evaluate previous history of diarrhea in family contacts.

The method of spread of these organisms within the family group, the relationship of familial infections to "relapses," and the role played by such infected families in the community problem of shigellosis are subjects of some importance that need further clarification. Most infected family members received specific therapy in this series. This approach to the problem needs further investigation.

SUMMARY

A review of the cases of shigellosis admitted to the pediatric services of Charity Hospital in New Orleans during 1955 revealed that this disease is still a problem at this hospital. The 62 cases reviewed represented 11 per cent of all patients admitted to the pediatric services with a diagnosis of diarrhea.

The clinical picture manifested by these patients was not distinctive enough to differentiate them from other types of diarrhea. Among the children with shigellosis there were two fairly distinct clinical pictures which could be correlated with the age of the patient.

The response of the patients to treatment was usually good and was apparently influenced more by supportive care, such as parenteral fluids and electrolytes, than by the type of chemotherapeutic agent or antibiotic used.

Over 50 per cent of the family contacts screened were found to harbor the same type of Shigella organism as the patient. Questions arise concerning the problem of familial infection, in relation both to a given case and to the community at large.

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DISORDERS OF THE APPENDIX; SOME UNUSUAL CASES *

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The purpose of this paper is simply to point out a variety of unusual conditions observed in the appendix in the past few years. Regardless of the underlying condition, inflammation is the process which calls clinical attention to the appendix. As will be seen, then, the cases to be presented all show appendicitis. In practically all cases of appendicitis there are essentially two underlying factors: The first is infection which is present in every case of appendicitis. The second is obstruction which is present in most of the cases of severe acute appendicitis.

I. NEOPLASMS

A. *Primary adenocarcinoma.*

The first case to be presented is that of a primary adenocarcinoma of the appendix. This patient was an eighty year old colored female who had pain in the right lower quadrant, vomiting, fever, tenderness and abdominal distention. A clinical diagnosis of appendicitis with retrocecal abscess was made and an appendectomy was performed. At operation the appendix was found to be ruptured and a pericecal abscess was also present. On pathologic examination of the specimen, it was found to measure 7 by 4 by 3 cms. There was abundant fibrinopurulent exudate over the entire external surface. It was noted on section that the lumen was occluded but it was only on histologic examination of sections that the occlusion was found to be due to a primary adenocarcinoma, projecting into the lumen and occluding it, (Fig. 1). The sections also showed a severe acute suppurative appendicitis. A right colectomy was suggested on the basis of the pathologic diagnosis of carcinoma of the appendix, but the patient refused further surgery. She was discharged from the hospital sixteen days after operation. She was readmitted one year later and an exploratory laparatomy at that time revealed involvement of the ileum and the anterior abdominal wall by the carcinoma. An ileotransverse colostomy was done and the patient discharged after thirteen days of hospitalization. This was three years ago. Nothing further is known of the patient at this time.

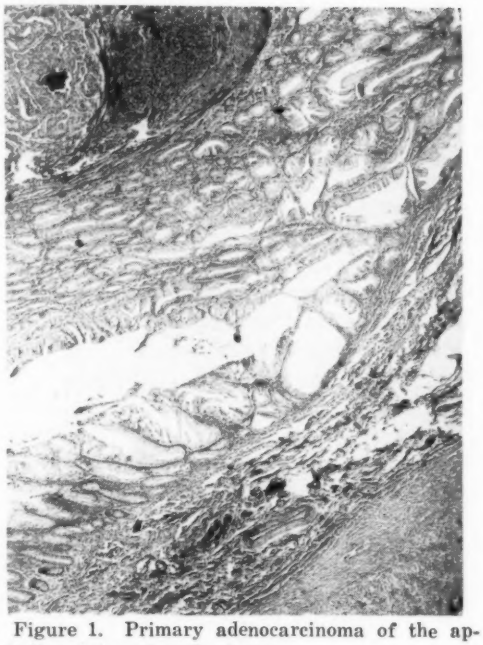

Figure 1. Primary adenocarcinoma of the appendix, obstructing the lumen.

In a study of 50,000 appendices, Collins [1] found 41 cases of primary adenocarcinoma, an incidence of 0.082 per cent. In general, adenocarcinoma of the appendix [2] is a disease of older people (55 years or over). The carcinoma is usually polypoid or fungoid, often at the base of the appendix, causing obstruction and subsequent acute appendicitis. It is to be noted that adenomatous polyps may occur in the appendix as they do in the colon.[3, 4] In fact, they may be a part of the picture of polyposis of the colon. It is highly probable that they also are premalignant lesions. They likewise may cause obstruction of the lumen of the appendix with subsequent appendicitis.

B. *Primary lymphosarcoma.*

The next case to be presented is that of a primary lymphosarcoma of the appendix. This patient was a male in his thirties with a clinical picture of typical appendicitis. An appendectomy was done and upon examination of the specimen there was evidence of acute suppurative appendicitis. On section, however, it was noted that the wall of the appendix was greatly thickened and it was found that the lumen appeared to be occluded by a bulge, extending beneath the mucosal surface. On microscopic examination, it was

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found that an extensive infiltration of lymphoid cells with hyperchromic nuclei involved large areas of the wall of the appendix (Fig. 2). It was clear that this involvement of the wall of the appendix by lymphosarcoma had caused obstruction of the lumen and subsequent acute appendicitis.

In Collins' series of 50,000 appendices,[1] the incidence of lymphosarcoma of the appendix is stated to be 0.014 per cent (7 in 50,000). Lymphosarcoma of the appendix may represent the primary site of involvement in this disease.[5] It may, of course, be part of a generalized lymphosarcoma. Untreated, it will surely become generalized, though it is known that at times lymphosarcoma in various portions of the gastrointestinal tract may remain localized for a time and surgical excision may result in apparent cure.

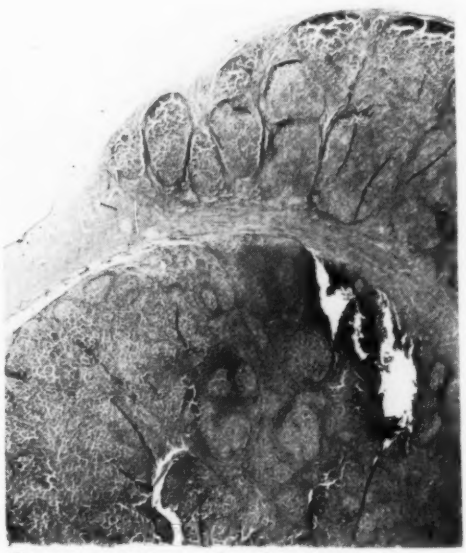

Figure 2. Primary lymphosarcoma of appendix, extensively involving the wall of the appendix, including the muscularis and serosa.

C. *Mucinous adenocarcinoma of cecum, invading base of appendix.*

The next case to be presented is that of a mucinous adenocarcinoma of the cecum with involvement of the base of the appendix. This patient was a 74 year old colored female who was observed three months previously with signs of intestinal obstruction. On the present admission there was pain and burning in the epigastrium with a palpable mass in the right lower quadrant. At operation a fungating mass was found in the cecum. The cecum and adjoining appendix were removed and on pathologic examination, it was found that the mass invaded the base of the appendix. The appendix was markedly dilated and contained mucoid material, typical of mucocele of the appendix. In the wall of the base of the appendix, the carcinoma took the form of an infiltrative adenocarcinoma (Fig 3). In other areas, the carcinoma was less well differentiated, having the appearance of a mucinous carcinoma.

Figure 3. Adenocarcinoma of cecum invading the base of the appendix, obstructing the lumen and causing dilatation (mucocele) of appendix.

D. *Carcinoids.*

The first of two cases of carcinoid to be presented is that of a 69 year old white female with a clinical diagnosis of appendicitis. An appendectomy was done and the appendix showed a typical acute suppurative appendicitis with cystic dilatation of the distal end. On section it was found that the proximal end was markedly thickened and showed both fibrosis and a diffuse infiltration of neutrophiles. In the dilated distal end of the appendix, within the wall, there were nests of cells, typical of a carcinoid. It is probable that this carcinoid was an incidental finding, the appendicitis being related to the fibrosis and partial obstruction in the proximal portion.

The more typical gross appearance of carcinoid is shown in the following case. This patient was an adult female with a clinical diagnosis of acute appendicitis and in whom the appendix showed a bulbous distal end with obliteration of the lumen

by the massive infiltration of neoplastic cells. These are seen microscopically in Figure 4. In this appendix also, there was a severe acute inflammatory process.

In the series, previously referred to, Collins[1] found an incidence of carcinoids of 0.522 per cent. Carcinoids may remain localized or occasionally may metastasize. About one in five will undergo metastasis, perhaps only to the regional lymph nodes or more rarely to distant organs. Unfortunately, the histologic appearance of those which metastasize and those which remain localized may present no differences by which the pathologist is able to predict the metastasis.

E. *Neurofibroma.*

The next neoplastic condition which was found to cause obstruction of the appendiceal lumen was a neurofibroma. This patient was a 34 year old colored female who had had an incision and drainage of an appendiceal abscess three months prior to the present admission. The abscess recurred and the patient was readmitted. Again incision and drainage was performed. Eleven days later an appendectomy was done. At the same time, a small mass was removed from the wall of the cecum. On pathologic examination, the appendix was found to be retort-shaped, measuring 7 cm. in length with a diameter of 2 cms. in the distal end and 1 cm. in the proximal end. Fibrinous exudate was present on the external surface. The lumen was found to be obstructed at the junction of the proximal third with the distal two-thirds, by grayish yellow tissue. The separate mass from the wall of the cecum measured 1 to 2 cms. in its various dimensions and was firm and grayish white. On section, the obstruction of the appendiceal lumen was found to be caused by whorled irregular fibers and intervening cells. A special stain for nerve fibers showed this tumor to be of neural origin. The cells, in this stain, are noted to be finely granular (Fig. 5). Sections of the tumor from the cecum showed a similar histologic appearance. The final pathologic diagnosis was "granular cell neurofibroma".

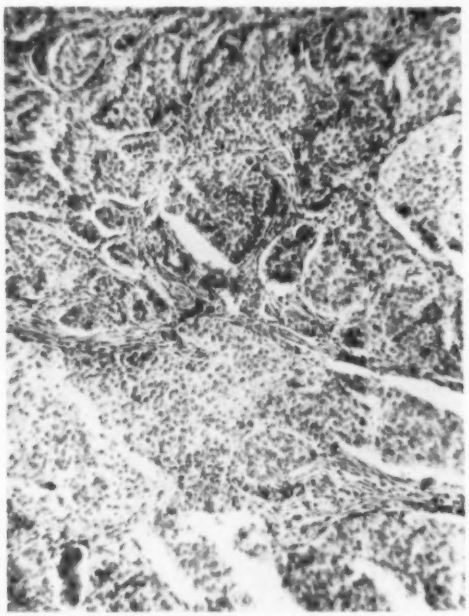

Figure 4. Masses of carcinoid cells, obliterating lumen of appendix in bulbous distal end.

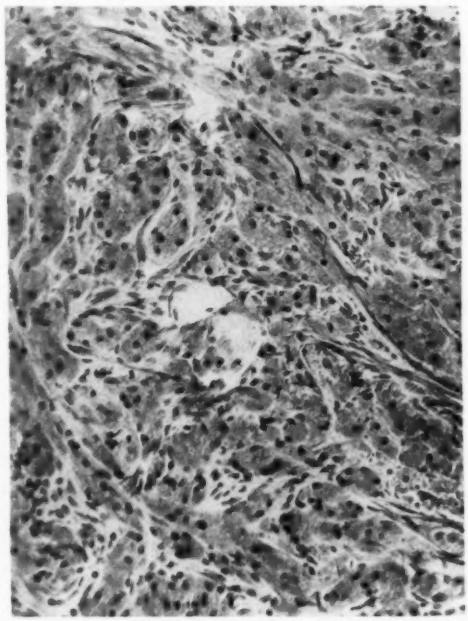

Figure 5. "Granular cell" neurofibroma of appendix, special stain to show details of the neural elements.

The incidence of neurofibromas of the appendix was found to be 0.38 per cent in Collins' series.[1] Not infrequently, one will find neuromatous proliferation of nerve elements in appendices in which fibrosis has obliterated the lumen. Not infrequently, also, a proliferation of the nerve plexuses of Meissner and Auerbach is seen in healing appendices. These neural proliferations are not true neoplasms. The tumor in the present case, however, is considered to be a true neoplasm of benign nature.

II. DIVERTICULA OF APPENDIX

In the next group of cases, the pathologic change was essentially inflammatory but showed a condition not often ascribed to the appendix, namely, one or more diverticula. The clinical picture leading to the appendectomy in these cases is usually that of acute appendicitis. On gross examination of the appendix, there may be evidence of acute appendicitis, but, in addition, there is often marked thickening of the wall with small blister-like projections from the surface. These projections may occur anywhere along the length of the appendix, more on the mesenteric than the antimesenteric border.[6] A typical diverticulum is demonstrated in figure 6. The diverticulum which is most commonly seen is an acquired diverticulum and is false, with herniation of the mucosa through the muscularis. The herniation may occur at vascular hiati or may be the result of destruction of the muscularis by intramural focal abscesses during an episode of a previous appendicitis. These acquired diverticula may be multiple. Congenital diverticula of the appendix are extremely rare. Acquired diverticula were encountered in 1.38 per cent of the cases studied by Collins.[1]

Figure 6. Diverticulum of appendix, consisting essentially of a herniation through the muscularis. The wall is composed largely of mucosa and thickened serosa.

At least two important facts may be related to the presence of diverticula in an appendix: (1) The diverticulum may be the site of rapid perforation when a recurrence of appendicitis begins; (2) a diverticulum of the appendix may rupture during the quiescent phase of appendiceal disease and become a source of pseudomyxoma peritonei.[7] For these two important reasons, an appendectomy should always be done at exploratory laparatomy when the appendix is found to harbor diverticula.

III. APPENDICITIS IN A NEWBORN INFANT

The final case to be presented is unusual by virtue of the time of life at which it occurred. This patient was a female infant, one of twins, delivered by cesarean section, five years ago. The birth weight of this patient was 3 pounds 14 ounces. Incidentally, the other twin is living and well at the present time. The patient was noted to become occasionally cyanotic shortly after birth. Two days later, cyanosis was more troublesome and the patient had begun to grunt and to vomit. The temperature became elevated to 101.2° F. on the seventh day of life. The baby died on the ninth day of life. At autopsy, the body weight was 1610 grams (3 pounds 7 ounces). The abdomen was markedly distended. The peritoneal surfaces were covered with a diffuse fibrinopurulent exudate. The stomach was dilated. The fibrinopurulent exudate was found to be most marked in the right lower quadrant and the appendix was dark red and roughened. (Fig. 7). In addition to the abdominal findings, a small zone of hemorrhage was noted in the right lateral ventricle of the brain. There was also an early acute meningitis. This then was a

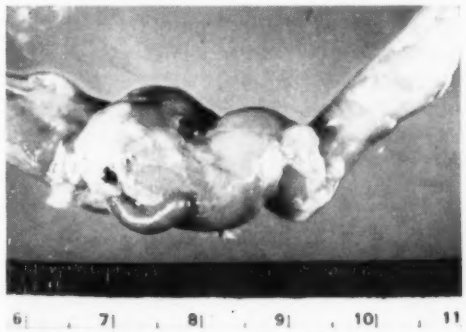

Figure 7. Appendix with attached cecum and ileum, showing acute appendicitis and fibrinopurulent exudate on peritoneal surface of adjacent structures.

case of acute appendicitis with peritonitis in a premature newborn infant of nine days.

In general, the diagnosis of appendicitis in young children is extremely difficult.[8] Often the total leucocyte count will not be elevated; the history is difficult to elicit or is atypical; and the physical findings may not be typical. In addition, young children frequently develop peritonitis rapidly following the onset of appendicitis.[8] Among the causes of this are the frequent use of cathartics in these children by ill-advised parents or other adults; the children, too, are unable to localize their complaints or to describe them; the underdeveloped omentum fails to protect and wall off the involved area; the high position of the cecum in children may be misleading to the clinician since the pain in the abdomen may seem to be unusually high.

SUMMARY AND CONCLUSIONS

1. A variety of neoplastic disorders, causing appendicitis by obstruction of the lumen has been presented: These include primary adenocarcinoma, primary lymphosarcoma, mucinous adenocarcinoma of the cecum, carcinoid, and neurofibroma.

2. Appendiceal diverticula result from previous inflammation of the appendix. They may lead to rapid rupture in recurrent appendicitis or they may perforate during the quiescent phase of appendiceal disease and become a source of pseudomyxoma peritonei.

3. A case of acute appendicitis with peritonitis occuring in a newborn premature infant is presented.

4. Regardless of the underlying pathologic condition in the appendix, it is an inflammatory reaction, appendicitis, which calls clinical attention to this organ.

Note: The author wishes to thank Dr. Joseph Ziskind for the use of the case of primary lymphosarcoma and Dr. Charles Dunlap for the case of neurofibroma.

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THE DEVELOPMENT OF VISION IN THE CROSS-EYED CHILD *

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Strabismus is not a rare condition. Surveys have shown that 1 per cent to 1.5 per cent of the children in a general population group are cross-eyed, and certainly each of you know of several such cases. Strabismus, squint, weak or paralyzed eye muscle, wall-eyed, cock-eyed, and cast in the eye are all synonyms for cross-eyes. The crossed eye may be turned laterally toward the temple, medially toward the nose, upward or downward.

MECHANISMS OF VISION

First, let us briefly analyze vision. There are two controlling mechanisms: (1) the extra-ocular muscles and their nerves which function to carry out the movements of the eyes; and (2) the sensory visual apparatus, which is responsible for the formation of images on the retina, the transmission of these images to the visual areas of the brain, and the blending of these bilateral images to form a single image with depth perception. These two delicately adjusted mechanisms are related to fixation, visual acuity, and fusion.

The individual first must be able to fixate each eye on the object he is trying to see because while the eyes are moving they do not see anything but large objects. They must stop and look steadily at

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smaller objects in order to see them. This process of holding the eyes steady is known as fixating. Secondly, each eye must have good visual acuity in order to see the smaller objects clearly. And thirdly, the individual must possess the ability to blend the images of the two eyes to form a single image. This ability is known as fusion.

The development of fixation, visual acuity, and fusion in a child was measured by Chevasse [1] and later confirmed by Gesell and his associates.[2] (Table 1) Their studies have shown that when a child is born he sees light but cannot follow it and does not have fixation. During the first few years of life the child must learn to see just as he must learn to walk and talk. Although he is born with two eyes, he must learn to use them. And like walking and talking, it is through practice, usage, and learning that he develops his abilities for fixation, visual acuity, and fusion.

When the eyes of a two week old infant try to fixate an object, they display only incoordinate disjunctive movements. At first these movements are inexact and the eyes simply swing pendulum-like until light strikes the fovea. By the end of the sixth week of life an infant is able to hold fixation for a few seconds. By a process of continuing adjustment for foveal fixation, an exact binocular fixation ability usually develops between the third and fifth months. The eyes fixate simultaneously and follow objects, and the child can reach for them fairly accurately, although his visual acuity is only 20/200.

If reflex fixation movements develop and become firmly established, the two eyes will remain parallel most of the time. Once this happens, vision begins to develop. The visual acuity is 20/150 at 12 months of age. The most rapid development of vision is during the first two years. At the end of that time the acuity is 20/45. The visual acuity reaches 20/20 between the fourth and sixth year.

The function of the fusion reflex is to correct any minor imperfections in the coordination of the two eyes. The fusion reflex is first recognized at the age of two years, at which time it is capable of complete extinction. By the age of three years these reflexes are becoming established, though they will suffer readily and seriously from disuse. At the age of four years the fusion reflexes are grounded, and while disuse may cause deterioration, it cannot cause extinction. Any loss sustained after this age can be rapidly overcome by re-use. The adjusting mechanism of the fusional reflex is capable of increasing its power of coordination and regulation with practice. Greater skill is

TABLE 1
STEPS IN THE DEVELOPMENT OF VISION *

Birth	Visual Acuity	Fixation and Stability
2 weeks		Aimless movements.
6 weeks		Momentary fixation.
5 months	20/2000	Fixation held for few seconds.
6 months	20/1000	Exact fixation held.
9 months	20/200	Normal field of fixation.
2 years	20/45	Fusion reflex is recognized. Capable of complete extinction.
3 years	20/30	Fusion reflex established. Deteriorates greatly from disuse.
4 years	20/25	Fusion reflex and visual acuity deteriorate from disuse; rapid recovery with re-use.
6 years	20/20	Fusion reflex and visual acuity more stable; may still deteriorate from disuse.
8 years		Fusion reflex and visual acuity unconditionally fixed.

* Modified from Lyle.[1]

thus acquired. By the age of five years the fusion reflexes have assumed unconditional fixity though they are still capable of some deterioration from disuse. After the eighth year they become unconditionally fixed and the period of instability has ended.

Thus during the first few years of life the child learns to see. His reflexes become perfected and thereafter the normal development of vision is dependent upon the continued use of both eyes. If for any reason during this developmental period, the child is deprived of the opportunity or is unable to practice and to learn the usage of his eyes, he most probably will become cross-eyed.

CAUSES OF CROSSED-EYES

The eyes may normally cross for a few seconds up to the age of 18 months. This is known as a physiological deviation and is due to incomplete development of the fixation process.

Some children have a wide epicanthal fold and may appear to be cross-eyed. This condition is known as pseudostrabismus, and frequently it is necessary to determine whether a true crossed-eye exists.

The most important thing in any cross-eyed case is to determine why the eye has crossed. The crossed-eye is only a symptom of the underlying condition which prevented the child from being able to use his eyes properly. Let us remember that there are three potentially lethal conditions which may be associated with crossed-eyes. In each of these the squint may be the first evidence of the disease.[3] They are: (1) brain tumor, (2) retrobulbar tumor, and (3) retinoblastoma. The latter is a highly malignant tumor of childhood, in which the eye shows a tendency to cross even before the characteristic gray, cat-like retina can be seen shining through the pupil.

There are a number of other conditions in which crossing of the eyes is present at one stage or another of the pathologic process and in which loss of vision may be the end result.[3] Among these diseases are:

1. *Retrolental fibroplasia.* The crossing of the eyes may be a late manifestation.

2. *Congenital cataract.* The squint is occasionally the first manifestation. This condition which is often associated with nystagmus may be monocular or binocular.

3. *Congenital muscle anomalies.* Strabismus fixus is an illustration.

4. *Errors of refraction.* Loss of vision may result from nonuse of the affected eye. If fusion develops normally, unequal refractive errors may not cause strabismus, because normal fusion can hold the eyes in alignment. If fusion is subnormal, or if it has not developed, crossing of the eyes may result.

5. *Monocular blindness from any cause.* If there is any latent muscle imbalance present the blinded eye will usually turn, since fusion no longer operates to hold the eyes parallel.

The muscular dysfunctions are the most common cause of crossed-eyes[3] and they are due to a variety of causes:

1. *Congenital weakness of the muscles.*

2. *Hereditary strabismus.* This may be inherited by the child even when it does not appear in the parents.

3. *Loss of muscle tone from general disability.* In such cases the squint tends to be periodic, disappearing when the child is in good health and emotionally stable and reappearing when he is ill, nervous or tired.

4. *Childhood diseases.* It is not uncommon to observe crossing of the eyes for the first time after measles, influenza, mumps or some other illness. In such instances the effect of the disease is merely to hasten the appearance of the squint. The latent weakness is already present and would have appeared sooner or later. This is not true of diphtheria which affects the eye muscles directly.

5. *Trauma (Birth injury).* This may occur during delivery and may become manifest then or occur during the growth

of the child. It may be the result of improperly applied forceps, but most often it is the outcome of spontaneous delivery. It has been estimated that in from a third to a half of all spontaneous deliveries the inevitable compression of the head during the process produces multiple tiny bleeding points within the brain. These minute hemorrhages, as a rule, are quickly absorbed. If, for any reason they are not, it is conceivable that some cerebral disturbance may occur, which may result in permanent damage and crossed eyes.

In search for the underlying factor responsible for the crossing of the eyes, it must never be forgotten that more than one cause may be present.

DOUBLE VISION

Frequently the question arises about how does a cross-eyed child see and does he see double. The answer depends on how old the child was when his eyes crossed, and secondly, what caused them to cross because as previously stated, a crossed eye is only a symptom of some underlying pathological process.

If the crossed eye has its onset after the age of six years and there is good vision in each eye, the child will see double and seek relief. When the crossed eye occurs prior to the age of six years and is due to a muscular dysfunction as most of them are, the child does not see double, but he learns to suppress constantly with his weaker eye. He develops abnormal habits of vision and these will become permanent if neglected. The longer they are allowed to remain the more difficult they are to change. The suppression in the weak eye becomes deeper and its visual acuity deteriorates, perhaps to less than 20/200 from disuse. This deterioration may be to the extent that the weak eye has a poor ability to fixate. This is one of the more important complications of leaving the cross-eyed child untreated.

DEVELOPMENT OF DETERIORATION

Actually if treatment is delayed and if obstacles to good vision are not corrected, the extra-ocular muscles will develop irreversible changes which alter their action and limit the motility of the eye. The muscle on one side of the eye will become hypertrophic and fibrotic, as a result of constant contraction and spasm. The antagonist muscle on the opposite side of the eye becomes thinned, atrophic and weakened, as a result of constant relaxation. And these muscular changes are another important complication of leaving the cross-eyed child untreated.

The cross-eyed child knows that he is different from other children. At first he attempts to conceal his abnormality by special positions of the head. In the end, he may become a personality problem, with manifestations ranging from lack of group participation to frank exhibitionism. The personality manifestations may become the most important complication of leaving the cross-eyed child untreated, and may actually be more serious than the squint itself.

THERAPY

The therapy of crossed eyes must obviously begin with an identification of the causes, since they will identify the form which treatment must take. Therapy must be directed at the parents even before it is applied to the child. Parents must be made to understand that the problem is one of visual function as well as one of appearance. The chances of a spontaneous cure, unless the crossing of the eye is of nervous origin, is practically nonexistent. Because the development of vision occurs early in life and is completed by the sixth to eighth year, there is no justification for the general belief that the correction of the squint may safely be deferred until the child is of school age, or even later.

The treatment is aimed at establishing good vision in each eye, good motility in each eye, and proper alignment of the two eyes as early in childhood as possible. The therapist must destroy abnormal visual habits and teach the child normal habits in an effort to restore normal development.

The progress of treatment is comparable to that of straightening teeth—a

long period of treatment with periodic adjustments. The child must be kept under observation during the period of visual development and until the age when the development of visual habits has become complete and permanent. There is no rapid cure for crossed eyes. If the child has a refractive error it should be corrected with glasses so that he will have the maximum opportunity to develop his vision. In teaching the child normal habits, it is often necessary to wear a patch over his better eye for a period of several months so that he will develop better visual acuity and motility in his weaker eye. It is often necessary to prescribe atropine in one eye to discourage the child's preference for using it. Orthoptic exercises may be prescribed to teach and develop a good quality of fusion so that the two eyes will become welded in their position of proper alignment. Surgery may be necessary to restore proper alignment and motility.

Whatever surgical or nonsurgical measures may be applied, the goal of therapy is to eliminate potentially fatal conditions, avoid personality disturbances, and establish stable habits of good binocular vision.

It is essential that the treatment of strabismus be begun before the potentials of normal binocular vision are lost. If treatment is deferred until after six years of age, the results will probably be limited to cosmetic improvements. Although this falls short of the ideal, it is of course a considerable gain because of the social and economic problem caused by being cross-eyed.

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STAPHYLOCOCCAL SEPTICEMIA
MANIFESTATIONS AND TREATMENT *

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The mounting importance of staphylococcal infections is illustrated by the statistics of deVries and Pritchard [1] who reported on cultures taken at autopsy at the Montreal General Hospital. Material was obtained from the heart's blood, cut surface of the lungs, and other infected areas in 213 autopsies. Cultures yielded *Micrococcus pyogenes* in 87 cases of which all but 7 were obtained from the heart's blood or lungs or both. The magnitude of the problem is also reflected in the large percentage of resistant strains which have emerged since the treatment of the first patient with penicillin in 1941.[2] Spink [3] reported, in 1944, that 12 per cent of 68 cultures of coagulase-positive strains of staphylococcus obtained prior to the introduction of penicillin were found relatively resistant to this antibiotic. Since then, numerous [4-6] reports have indicated an increasing number of resistant staphylococci isolated. This is illustrated by Knight's series [7] from Bellevue Hospital where 65 per cent of the staphylococci isolated in 1953 and 1954 were resistant to chlortetracycline, oxytetraclycine, streptomycin, and penicillin. The strains showed intermediate degrees of sensitivity to chloramphenicol and nearly all were susceptible to erythromycin. From December 1, 1952, until March 1, 1954, Wise [8] and associates have observed 23 patients with infections caused by erthromycin-resistant staphylococci. In this paper, we are going to describe the findings and treatment of two cases of staphylococcal septicemia with pulmonary involvement.

CASE REPORTS

Case No. 1. This 22 year old white male was admitted to Tokyo Army Hospital on November 12, 1954, as a transfer from another hospital in Japan. The patient entered the latter hospital

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on October 11, 1954, with an illness of three days' duration characterized by vomiting, anorexia, and fever. Two days after admission, he developed a cough productive of mucopurulent sputum which was occasionally blood tinged. He was treated with antibiotics and the fever subsided. He was transferred to Tokyo Army Hospital for further diagnostic studies and treatment. The patient stated that he had had hepatitis in Korea in 1951 and again in July and August 1954.

Physical Examination: On admission, the patient was a well developed and slightly thin white male, who did not appear acutely ill. The blood pressure was 106/68. The temperature was 98.6° F. The pupils were equal and reacted to light and accommodation. Ophthalmoscopic examination was normal. The throat and tongue were normal. The chest expansion was equal bilaterally. The lungs were resonant and clear. The heart was not enlarged. There was a regular rhythm. There was a grade II systolic apical murmur. On examination of the abdomen, the spleen was palpable just below the left costal margin. The genitalia were normal. Rectal examination was normal. The biceps and patellar reflexes were physiological. There was no edema or significant lymphadenopathy.

Hospital Course: On admission to Tokyo Army Hospital, no antibiotics were started. On November 17, 1954, the temperature spiked to 104.6° F. Examination at that time revealed no nuchal rigidity. There were a few coarse rales at both bases. There was a grade III systolic murmur heard over the entire precordium. The abdomen, prostate, and lower extremities were normal. Two blood cultures were taken and treatment was immediately started with penicillin, 1,000,000 units every two hours. On November 18, the temperature spiked to 105° F. Marked tenderness was noted in the right upper quadrant of the abdomen. On November 19, the two blood cultures were reported hemolytic *Staphylococcus aureus*, coagulase-positive, which was sensitive to chlortetracycline, chloramphenicol, oxytetracycline, bacitracin, and erythromycin, but resistant to penicillin, streptomycin, polymixin B, and neomycin. Tenderness was still present in the right upper quadrant. Penicillin was discontinued and oxytetracycline was started, 500 mg. four times a day. On this therapy, the temperature returned to normal by November 20. Fever recurred on November 23, so chlortetracycline, 500 mg. four times a day, was added to the treatment. On this regimen, the temperature remained normal until December 1, when it went to 100.8° F. On December 5, it spiked to 102.8° F. Because of continued fever, it was decided, on December 11, that other antibiotics should be added to the regimen. So chloramphenicol, 500 mg. four times a day, erythromycin, 500 mg. every six hours, and penicillin, 1,000,000 units every two hours intramuscularly, were started. Chlortetracycline was stopped but oxytetracycline was continued. Subsequently, the patient became and remained afebrile. It was decided that since this was a staphylococcal septicemia, these medications should be continued for a period of at least two months. On February 16, 1955, the penicillin and erythromycin were stopped; on February 24, the oxytetracycline was discontinued; and on March 4, the chloramphenicol was stopped. There were no recurrences of fever. The patient was gradually ambulated and returned to duty by April 1, 1955.

Radiographic Studies: The chest x-ray of October 18, 1954, (Fig. 1), was a lordotic view

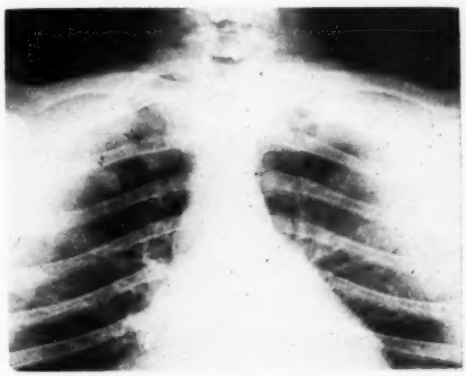

Figure 1.

which showed a soft area of infiltration located in the third right interspace in the mid-lung zone. There was a smaller area of infiltration in the third right interspace near the mediastinum. There was also a soft infiltration in the left apex with an area of increased radiolucency measuring almost 2 cm. in diameter which on laminograms was demonstrated to be a cavity. The x-ray of November 5 (Fig. 2) demonstrated decrease in the size of the apical cavity on the left and a thin walled cavity .75 cm. in diameter in the second anterior interspace on the left. There was also a patchy infiltration in the left lower lung field with obliteration of the left costophrenic angle. The right lung was clear except for marked increase in the bronchovascular markings at the base. The chest x-ray of December 6 (Fig. 3) showed a soft area of infiltration from the fourth anterior rib to the diaphragm with obliteration of the right costophrenic angle. The left lung was clear except for a soft infiltration in the fourth anterior interspace. The x-ray of December 9 showed some clearing of the previously described infiltration in the right lower lung field. The costophrenic angle was clear. The lateral view localized the infiltration to the right middle lobe. There was decrease in the density at the left base. The film of December 15 showed areas of linear infiltration in the right fourth anterior interspace. There was a .75 cm. soft area of infiltration in the third left anterior interspace.

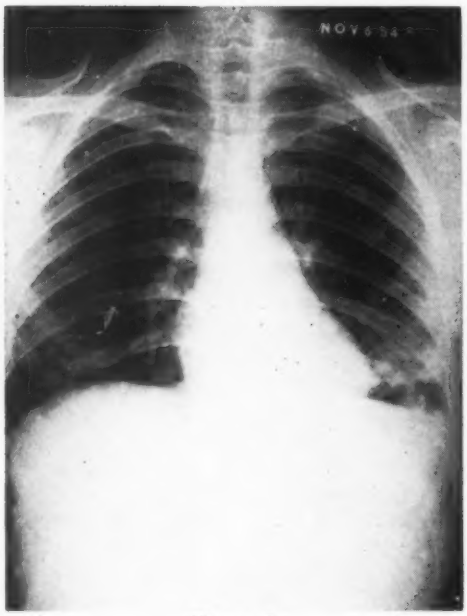

Figure 2.

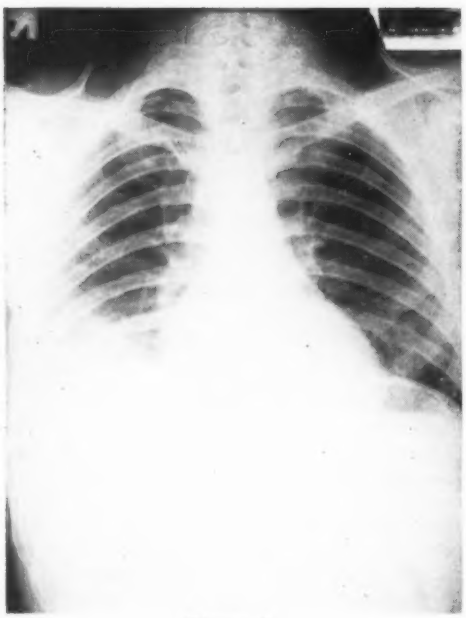

Figure 3.

The film of January 4, 1955, showed progressive clearing of the previously described densities. The chest x-rays of March 9 were normal.

Case No. 2. This 21 year old white male was admitted to Tokyo Army Hospital on August 11, 1955, as a transfer from another hospital in Japan. On April 16, 1955, he entered a hospital in Korea, with a history of cough, right chest pain, and malaise of twelve hours' duration. He gave a history of having taken intravenous morphine for two months prior to the onset of his illness. On admission to the Army hospital in Korea, his temperature was 102.8° F. and there were dullness and expiratory rales at the right base. Three blood cultures were positive for a staphylococcus. The patient was treated in Korea with multiple antibiotics and transferred to a hospital in Japan by June 10, 1955, where similar treatment was continued and finally he was evacuated to Tokyo Army Hospital. Just prior to transfer, he was receiving chloramphenicol and erythromycin.

Physical Examination: The patient was a well developed and well nourished white male. The temperature was 100° F; the pulse was 86 per minute; the respirations were 20 per minute; and the blood pressure was 120/80. There were no petechiae. The pupils were equal and reacted to light. Ophthalmoscopic examination was normal. The throat and neck were normal. Examination of the lungs revealed impaired resonance over the left base posteriorly with a pleural friction rub in this area. The heart was not enlarged. There was a regular rhythm. There was a grade III systolic murmur adjacent to the sternum in the fourth intercostal space. The abdomen and genitalia were negative. There was no edema or lymphadenopathy. The biceps and patellar reflexes were physiological.

Hospital Course: It was decided that chloramphenicol and erythromycin should be continued because of the apparent response. Chloramphenicol was given, 1 gram every six hours, and erythromycin was administered, 200 mg. every six hours. On this regimen, the fever was low grade in character until August 29, when the temperature spiked to 102.4° F. Five blood cultures were taken and the patient was started on penicillin, 1,000,000 units every two hours. All of these cultures were positive for a hemolytic staphylococcus, coagulase-positive, which was sensitive to erythromycin, streptomycin, chlortetracycline, chloramphenicol, oxytetracycline, bacitracin, and neomycin, and resistant to penicillin and polymixin B. On September 2, the temperature spiked to 103°, and on September 3 to 104°. At this time, streptomycin, 1 gram twice a day, and oxytetracycline, 500 mg. every six hours, were added to the treatment. On the combination of penicillin, chloramphenicol, erythromycin, oxytetracycline, and streptomycin, the patient became afebrile. On September 11, the oxytetracycline was discontinued, and on September 12, the chloramphenicol and erythromycin were stopped because of vomiting. On September 15, the streptomycin was reduced to 1 gram twice a day every second day. Erythromycin was restarted and penicillin was continued. On November 14, penicillin and streptomycin were stopped and on

November 21 erythromycin was discontinued. The patient remained afebrile and was returned to duty December 15, 1955.

Radiographic Studies: The chest x-ray of August 12, 1955, (Fig. 4) showed a confluent

Figure 4.

irregular area of infiltration in the lower third of the left lung which measured 3 by 5 cm. Just above this, there was a smaller lesion which measured 1 by 1.5 cm. In the right base, near the costophrenic angle, there was an ill defined infiltration measuring about 3 cm. in diameter. The x-ray of August 22 demonstrated that the area of pulmonary infiltration in the base of the right lung had cleared and there was a residual linear scar. The two areas of infiltration in the left lung showed some clearing. The film of August 29 showed the residual scar still present at the right base. There had been progressive clearing of the lesions at the left lung base. The chest x-ray of September 26, (Fig. 5) showed that the previously decribed densities in both lung bases had cleared except for slight linear scarring. A new lesion was present in the right upper lobe at the level of the first anterior interspace. There was present an area of increased radiolucency in the middle of this lesion which suggested cavitation. The film of October 20 showed almost complete clearing of the previously described lesions. The chest x-ray of November 29 was clear.

DISCUSSION

The pathogenicity [9] of the staphylococcus *(Micrococcus pyogenes)* can be determined by its reaction to the coagulase

Figure 5.

test. A loopful of bacteria from a twenty-four hour culture is added to 0.5 ml. of a human or rabbit plasma. This is incubated for three to eight hours at 37° C. A coagulum will be formed with the pathogenic strains only. The pathogenic strains produce an antigenic factor which, through the intermediary of a plasma activator, causes the conversion of fibrinogen to fibrin. It has long been taught that staphylococci which form orange colonies surrounded by a zone of hemolysis on blood agar plates are pathogenic. This is generally true, but highly invasive strains may appear as nonhemolytic white colonies. Bateriophage sensitivity has been utilized as an aid in differentiating the individual strains of staphylococci. Fisk [10] demonstrated that phages derived from coagulase positive strains did not produce lysis of coagulase-negative strains. Phage typing [11] has been used extensively in tracing the origin and spread of staphylococcal infections. For example, Hobbs [12] demonstrated that the staphylococci isolated from several cases of breast abscess were of the same phage type as those from the throat of a nurse who was a carrier.

The cases presented illustrate the pul-

monary findings that may occur in staphylococcal septicemia. The primary site in each is not certain. The second patient was a morphine addict and had a murmur compatible with a ventricular septal defect so that he could easily have had a bacterial endocarditis. The first case might have had a staphylococcal pneumonia as the original source of the septicemia, since his first symptoms were referable to the respiratory tract. Staphylococcal pneumonia is usually not primary but may complicate other acute or chronic respiratory disease. Finland [13] reported a group of staphylococcal pneumonias which occurred in an influenza epidemic. There are other clinical pictures which can arise in staphylococcal disease besides the classical abscesses which occurred in our patients. Spink [9] has emphasized that certain strains elaborate an exotoxin which can cause nausea, vomiting, diarrhea, cyanosis, jaundice, convulsions, respiratory and circulatory failure. He has reported a case of a healthy young adult who died within fifty-one hours from an infected laceration. Certain strains can also produce a dermonecrotoxin which may cause extensive tissue necrosis. We are all familiar with the enterotoxin which results in staphylococcal food poisoning.

TREATMENT

In managing the severe forms of staphylococcal sepsis. several procedures should be followed. Before treatment, one should obtain adequate material in the form of blood, sputum, or pus for culture and sensitivity studies. As soon as this is done, treatment should be immediately instituted. Penicillin is still the most effective agent in the treatment of sensitive staphylococci. For this reason, initial treatment of severe infection should always include penicillin. The dosage should be adequate. Spink [9] advocates 500,000 units every four hours intramuscularly and, in addition, 500,000 units intravenously in 10 ml. of saline every twelve hours for the first forty-eight to seventy-two hours. The first two patients were given 1,000,000 units every two hours, intramuscularly. Spink also feels that this initial treatment should include another agent. He recommends bacitracin in patients with good renal function in dosage of 20,000 units every six hours. Since bacitracin is nephrotoxic, in the patients with seriously impaired renal function he advocates erythromycin in dosage of 2 to 3 grams a day. Once the sensitivity studies have returned, you can switch to a combination of antibiotics which may best fit these studies. Fisher [14] and associates have reported on 38 cases of staphylococcal endocarditis which occurred from 1933 through 1953. Of the group, 13 occurred from 1950 through 1953, and of these 54 per cent recovered. An interesting fact is that during this period when other investigators were reporting penicillin resistance, most of the organisms isolated were sensitive to this antibiotic. In Fisher's cases, no patient was cured whose treatment did not include penicillin. Three of Fisher's patients were found to be resistant to less than 12.5, 62.5 and 50 units of penicillin respectively, yet massive doses of penicillin plus erythromycin led to favorable outcome in each. One of these did not respond until penicillin was added. For these reasons, Fisher and his associates feel that regardless of the laboratory findings, penicillin should always be included in the therapeutic regimen. In this we agree, providing that the patient is not allergic to this drug.

A new antibiotic has become available since the treatment of our patients, which seems to be quite effective in staphylococcal infections. It is novobiocin which has been isolated from cultures of the actinomycete, *Streptomyces niveus.* [15] .The recommended oral dosage for infections of moderate severity is 500 mg. every six hours. The most common side reactions reported have been allergic dermatitis, which may be associated with fever.[16] Mild diarrhea has occurred. Thus far, no damage to the kidneys or liver has been reported. Martin [17] and associates noted two cases of leukopenia but differential

counts remained normal and the abnormality did not persist with continued administration of novobiocin. David and Burgner [18] reported two cases of mild neutropenia. This subsided with discontinuance of the drug. Another new antibiotic, spiramycin [19] is reported to be effective in most strains of resistant staphylococci.

The third and very important step in the treatment of staphylococcal infections is the adequate surgical management of all suppurative and necrotic tissues or cavities. If this is not done, it will contribute to the development of resistant organisms and serve as a constant source of infection. The fourth thing to remember in the treatment of staphylococcal infection is prolonged therapy. Once a staphylococcal septicemia is under control, treatment should be continued for at least six to eight weeks longer because fatal relapse may follow early cessation of treatment.

It should be mentioned that although short courses of adrenocorticotropic hormone and corticosteroids have been advocated [20] for certain critically ill patients with various infections, they should only be used in staphylococcal disease with very adequate antibiotic coverage. These hormones decrease the inflammatory reaction and thus might increase the chance of spread of a staphylococcal infection. Fisher [14] has emphasized the danger of concomitant or prior treatment with these drugs in staphylococcal infections.

In conclusion, it should be emphasized that unless all of us are more conservative in the unnecessary use of antibiotics, more and more resistant bacterial strains will result. Erythromycin and novobiocin should be reserved for the more resistant staphylococci. Since the hematologic complications of chloramphenicol were demonstrated a few years ago, its decreased use may make it also available in the treatment of the more resistant organisms.

SUMMARY

Two cases of staphylococcal septicemia with pulmonary involvement and successful treatment are reported. The bacteriology and symptomatology of staphylococcal infections have been briefly reviewed. It is emphasized that treatment of severe staphylococcal infections should include four procedures. Before treatment, adequate culture material should be obtained. Immediate massive antibiotic treatment is advocated. Surgical management of all suppurative and necrotic tissues or cavities should be instituted. Once a staphylococcal septicemia is under control, treatment should be continued for at least six or eight weeks. The danger of corticotropin and the corticosteroids in staphylococcal disease has been emphasized.

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COMPULSORY SOCIAL SECURITY INSURANCE COVERAGE FOR PHYSICIANS VERSUS THE JENKINS-KEOGH BILL

In the coming session of Congress and its usual tumult, there will be consideration again of compulsory Social Security insurance coverage for physicians, and also, of the more desirable Jenkins-Keogh bill. These bills will represent two opposite points of view in which all physicians are vitally interested.

A satisfactory sampling of opinion among the doctors of the nation has indicated that the majority, in the proportion of approximately 3 to 2, are opposed to this additional tax. The reasons why physicians should oppose the tax have been discussed in these columns [1, 2] before and have been comprehensively covered in articles of *The Journal of the American Medical Association*.[3-5]

After due consideration is given to the facts of the Social Security insurance program, it is seen that the majority of physicians have due cause to oppose it. To paraphrase the comment of a famous French diplomat who is reported to have said—that the Holy Roman Empire was neither holy, nor Roman, nor an empire—the Social Security insurance program is neither social, nor secure, nor insurance. It is not social because it is primarily political. It is not secure because it is subject to any action by any Congress every two years; and it is not insurance because the individual covered receives benefits according to the provisions of an inherently political Congress, and not according to the equity of his payments.

According to the estimate of actuaries, the Social Security program has already assumed obligations equal to the national debt of the United States. The receipts under the operation of the law, at present, are estimated as 6.7 billion in contributions and 500 million in interest. The expenditures will be 6.1 billion. The trust fund exists only as bonds of the United States government. Both the contributions and the bonds are supported only by taxes. The income and the outgo are almost balanced now, before the new provisions and expensive commitments have had their effect and at a time when we are experiencing our highest taxation and greatest prosperity in history. In any period of national economic depression of major degree, the demands for Social Security money will go up and the sources of income will go down. This can only be met by increase in taxes, which would be politically unpopular and economically depressing. The obvious adjustment is that a Congress influenced primarily by politi-

cal considerations will make reduction in benefits. On the other hand, the plans of real insurance are made in such a manner as to anticipate and meet just such contingencies in their contractual responsibilities.

In a masterful analysis of Social Security insurance law, Webster and Coffey [4, 5] have shown that in the insurance market the physician can secure for himself, in comparison with the most favorable applications of the Social Security law, far better coverage. Commercial insurance would provide from two to nine times more protection, than he would have under the law. These figures do not agree with those given by a certain group within the profession, who wish for compulsory Social Security for doctors.

It is clear from the operations of the law that physicians in the older age groups—who, incidentally, have been those who opposed compulsory inclusion of doctors—are the ones who would benefit the most from this type of pension coverage, and that those who have been most anxious to include physicians are in the younger groups who would stand to lose the most in their long years of payment. Since six out of seven self employed physicians do not retire, the tax imposed upon them by compulsory coverage would be unjust and unreasonable. Since the average age at death of physicians is just under seventy, only a few would be able to enjoy full benefits of the law's provisions while still working at 72.

The Jenkins-Keogh bill would if enacted provide insurance coverage relieving a tax injustice and would meet the needs of physicians far better than compulsory coverage under the law. The effect of its operation would allow the individual to set aside money for pension purposes in the years of his greatest earnings, and pay tax on these earnings in the years of his declining income. At the present time, millions of individuals in industry are enjoying what amounts to this considerable tax advantage. In other words, Keogh states that in our existing federal tax laws there is an inequity and discrimination against self employed individuals which cannot be denied. Enactment of the Jenkins-Keogh bill would bring about a situation in which it would no longer be an economic sin to be self employed. This bill in the past has received lip service from many individuals in Congress from whom support has been sought, and it then dies in Committee. The objections to it are wordy but may be simplified in two statements: It is estimated it would cause a loss of approximately 150 million dollars in annual income tax revenue; and the other is that it would be doing the physicians a favor.

The answer to the first objection is that a mere 150 million dollars is a drop in the bucket compared to what a prodigal government wastes in improvident welfare schemes over the globe. Even if such a loss was significant this money is being unjustly squeezed from the taxpayers supplying it. The second objection has no answer.

The prospects of the Jenkins-Keogh bill passing this year are better than formerly. The American Bar Association has undertaken a vigorous campaign, with which it is of great interest to physicians everywhere to cooperate and support by all means possible. The benefits of the law will accrue to many million self employed besides the 160 thousand doctors, who have been advocating such provisions.

The coming Congress, therefore, represents an opportunity to the physicians to make clear their opposition to compulsory Social Security insurance coverage and their support of the equitable and desirable Jenkins-Keogh bill.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

INTERESTING FACTS FURNISHED BY THE A.M.A. WASHINGTON OFFICE

Part I

(July 1, 1956, to June 30, 1957)

FEDERAL MEDICAL-HEALTH SPENDING FOR FISCAL YEAR 1957

This is our fourth annual report on the Federal medical budget. To the extent we can make it so, it is a factual, objective study of how much the U. S. Government is spending this fiscal year in all health and medical fields. We are dealing with money that has been appropriated and currently is available to be spent. This SPECIAL REPORT is based on the actual appropriations, and on information obtained directly from Federal agencies and departments.

When talking in billions of dollars, it is often difficult to make the totals meaningful. However, here are some conclusions:

1. What the U. S. is spending in health fields alone represents an average cost of $15.17 per man, woman and child.* Incidentally, it is costing each of them $1.78 more this year than last.
2. If only wage-earners are considered,* they will be paying on the average $38.72 each to finance the Federal government's health-medical operations. That is $4.40 more than they paid last year.
3. The average family * will be paying $54.61 this year for the U. S. government's health-medical activities.
4. Even in an overall Federal budget of $61.2 billion, the total health cost is not insignificant. It is a billion dollars more than the cost of running the Commerce Department, half a billion more than all Agricultural Department expenses and six times Interior Department's budget.
5. Mostly because of spectacular increases for research, health programs of the Department of Health, Education, and Welfare this year will cost half again as much as they did last year.
6. For the first time since World War II, medical costs of Veterans Administration top the list, passing the Defense Department. A close third is the Department of Health, Education, and Welfare.

This SPECIAL REPORT is based on spending alone. We do not undertake to evaluate the many individual programs. Obviously many of them are necessary just to maintain this country's unsurpassed public health standards. Others undoubtedly will pay rich dividends in procedural and scientific discoveries. Some probably could be questioned But the only purpose of this study is to show exactly what they cost.

* Population, family-size and employment figures from Census Bureau.

MEDICAL-HEALTH BUDGETS OF FEDERAL DEPARTMENTS AGENCIES AND COMMISSIONS FOR THIS FISCAL YEAR

VETERANS ADMINISTRATION

(This Year: $825,024,300 — Last Year: $790,185,800)

In-Patient Care in VA Hospitals $619,614,000

(Last year: $615,869,000)

VA's largest single medical appropriation covers in-patient care in 173 VA hospitals and provides for an average of 121,865 beds a day. At present VA reports daily patient load at 111,540, just over 91% of capacity. The appropriation includes salaries of physicians and other personnel, medical rehabilitation of veterans, dietetic and nursing services, social services and special services, such as recreation and transportation of veterans.

Out-Patient Care $82,638,000

(Last year: $85,471,200)

The bulk of this appropriation is for out-patient care in about 100 VA clinics. The remainder is earmarked for fees to physicians ($7,738,000) and dentists ($8,415,000) under the home-town care program.

Modernization and Replacement Construction $51,635,000
(Last year: $30,000,000)

This amount is for work on existing units costing over $250,000 a project, and equipment for new units. The money is available until expended. (See Major Alterations and Improvements item below for smaller projects.)

Domiciliary Care $29,319,000
(Last year: $23,062,500)

Domiciliary care is being provided in 17 VA facilities for about 17,000 veterans who, while incapacitated for employment, are not in need of full hospital care. VA also makes payments to 30 state homes in approximately 30 states with a daily patient load of around 9,000. Payments this fiscal year are estimated at $5,888,000 (under a new law, P. L. 613, 83rd Congress, Federal contributions to these homes were raised from a maximum of $500 to $700 a year per patient).

Contract Hospitalization $13,967,000
(Last year: $15,237,300)

This appropriation finances an average daily patient load of 3260 veterans in Federal hospitals other than VA and in state and municipal hospitals. Patients in Federal non-VA hospitals are estimated at 1345 and in non-Federal hospitals, 1915. Psychotic cases make up the largest single category of contract cases.

Medical Administration $7,300,000
(Last year: $7,422,000)

To operate the VA Department of Medicine and Surgery in the Washington central office and the seven area medical offices; included are salaries, travel and like expenses.

Medical Research $10,990,300
(Last year: $ 6,381,600)

For research, mostly in VA hospitals. The breakdown: general medical and surgical research, $5,870,000; atomic medical research, $2,000,000; prosthetics testing, $990,300; neuropsychiatric, $1,250,000; tuberculosis, $780,000; other, $100,000.

Major Alterations, Improvements and Repairs $4,533,000
(Last year: $3,900,000)

For alterations, improvements and repairs to VA clinics and domiciliaries (costing less than $250,000 per project).

Supply Depot Operations $1,628,000
(Last year: $1,642,200)

For maintaining and operating supply depots handling the purchase, shipping and storage of medical supplies and equipment used by the Department of Medicine and Surgery.

Medical Education and Training $1,400,000
(Last year: $1,200,000)

For VA training programs for physicians and other VA personnel, in medical specialties and auxiliary services.

Medical Care—Philippine Veterans $2,000,000
(Last year: not reported)

Until 1960 the U. S. Government will contribute for the care of Philippine veterans.

DEPARTMENT OF DEFENSE

(This Year: $790,105,000 — Last Year: $818,104,500)

**Army Medical Services* ..... Approx. $290,000,000
(Last year: $300,000,000)

The estimated cost includes expenses normally associated with the operation of military hospitals and dispensaries, military and civilian salaries, medical supplies and equipment, utilities, communications, transportation, travel subsistence, maintenance and repair of buildings and grounds, expenses for construction, medical education and training, medical research and preventive medicine.

**Air Force Medical Services* ..... Approx. $223,000,000
(Last year: $286,000,000)

The estimated cost includes expenses normally associated with the operation of military hospitals and dispensaries, military and civilian salaries, medical supplies and equipment, utilities, communications, transportation, travel subsistence, maintenance and repair of buildings and grounds, expenses for construction, medical education and training, medical research and preventive medicine.

**Naval Medical Services* ..... Approx. $277,000,000
(Last year: $232,000,000)

The estimated cost includes expenses normally associated with the operation of military hospitals and dispensaries, military and civilian salaries, medical supplies and equipment, utilities, communications, transportation, travel subsistence, maintenance and repair of buildings and grounds, expenses for construction, medical education and training, medical research and preventive medicine.

Office, Asst. Secretary of Defense (Health & Medical) ..... Approx. $105,000
(Last year: $104,500)

For salaries, travel and administration of this office, and for travel expenses and consultant fees for the Defense Department Civilian Health and Medical Advisory Council.

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* All figures are estimates because appropriations are not broken down into categories of medical expenditures. Anticipated reduction in the Department of Defense medical program during Fiscal Year 1957 is primarily attributable to more effective joint utilization of the facilities, fewer personnel assigned to operation and a lower level of hospital and dispensary construction. Reductions, however, have been partially offset by an estimated expenditure of $41 million during Fiscal Year 1957 for the Dependent Medical Care Program.

DEPARTMENT OF HEALTH, EDUCATION AND WELFARE
(This Year: $772,661,800 — Last Year: $526,935,400)

Division of Hospital Facilities $126,381,000
(Last year: $112,250,000)

This division administers the Federal aspects of both the original and expanded Hill-Burton programs. The total appropriation is divided into the following four categories:

Hill-Burton Original Program $102,800,000
(Last year: $ 88,800,000)

This appropriation, allotted to the states on the basis of population and per capita income, assists in the financing of new hospitals and related health facilities construction under the original Hill-Burton program. To date Federal funds have partially financed approximately 2,869 projects, including nearly 132,096 hospital beds, and 636 public health centers, 18 state health laboratories and approximately 100 nurses homes and training facilities. To date Federal contribution represents $762,000,000 and is matched by sponsors' funds amounting to $2,370,000,000.

Medical Facilities—Category Program $21,000,000
(Last year: $21,000,000)

The total allotted to the states this year on a population-per capita income formula will assist in the financing of new construction under the 1954 amendments in four categories as follows: $6,500,000 for hospitals for the chronically ill and impaired; $6,500,000 for diagnostic centers, or diagnostic and treatment centers; $4,000,000 for nursing homes; and $4,000,000 for rehabilitation facilities. As under the original program, the Federal share may range from one-third to two-thirds of the total project cost. As of July, 1956, there was approved a total of 204 projects divided among the four categories as follows: Facilities for chronically ill, 42; Diagnostic-treatment centers, 77; Nursing homes, 42; and Rehabilitation facilities, 43.

Hill-Burton Administrative Expenses $1,381,000
(Last year: $1,250,000)

This appropriation is used for salaries and expenses for the hospital survey and construction program for Washington, D. C., and eight regional offices at the Federal level.

Research $1,200,000
(Last year: $1,200,000)

Authorized in 1949 but not appropriated until 1955 was this item for research, experiments and demonstrations on utilization of hospital services, facilities and resources. The bulk of the money is earmarked as grants to states, universities, hospitals, hospital associations, professional associations, and community organizations and a small amount for direct research by Public Health Service.

(Continued in January issue)

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Independence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

COURSES IN THE TREATMENT OF ACUTE TRAUMA AVAILABLE TO PHYSICIANS

It has come to our attention that the Army Medical Service will conduct courses in "Surgery in Acute Trauma" at the Brooke Army Hospital, Brooke Army Medical Center, Ft. Sam Houston, Texas, May 6-8, 1957, and the William Beaumont Army Hospital, Ft. Bliss, Texas, April 1-3, 1957. Similar courses will be held in other parts of the United States, but no others here in the Southwest. Civilian physicians interested in attending either of these courses may apply direct to the Commander of the Hospital where the course is to be conducted.

Those who have attended courses similar to these, given at Walter Reed Medical Center, Washington, D. C., and at Brooke Army Hospital, Ft. Sam Houston, Texas, cannot speak too highly of them. It is expected they would be of great interest to all physicians called upon to treat injuries, and to all those with Civil Defense responsibilities.

SURGICAL HISTORY VOLUMES APPEAR

The fourth volume in a series of 14 on the history of surgery during World War II appeared in October to be followed by an additional volume before the first of the year. The surgical volumes are part of the 40 odd volume work entitled Medical Department, United States Army in World War II. Editorial office for the surgical volumes of the medical history is in Tulane School of Medicine.

The title which has just appeared is Volume I on General Surgery which covers shock, resuscitation, anesthesia and abdominal injuries, and has a final chapter on the closure of colostomies. The title about to appear is Orthopedic Surgery in the European Theater of Operations.

Chairman of the Advisory Editorial Board for Surgery is Dr. Michael E. DeBakey, formerly Associate Professor of Surgery, Tulane University School of Medicine, and now chairman of the Department of Surgery, Baylor University College of Medicine. Associate editor is Miss Elizabeth M. McFetridge.

Other surgical volumes which have appeared are on the physiologic effect of wounds, published in 1952, and hand surgery and vascular surgery, both published in 1955. Three surgical volumes now in press or in preparation for production are Orthopedic Surgery in the Mediterranean Theater of Operations, Ophthalmology and Otolaryngology, and Cold Injury. This last is believed to be the most inclusive discussion of this type of trauma ever to be published. Two volumes on neurosurgery are now in active preparation and are expected to be ready for publication in 1957.

The entire 40 odd volume series prepared under the direction of Major General S. B. Hayes, the Surgeon General, United States Army, will contain in addition to the surgical volumes, 7 on preventive medicine, 4 on internal medicine,

2 on neuropsychiatry, 8 on administrative subjects, and the remainder on miscellaneous subjects. The volumes published to date include 1 administrative volume, 2 preventive medicine volumes, and a dental volume, in addition to the 4 surgical volumes.

The Government Printing Office is publishing the entire work.

POSTGRADUATE COURSES ON DISEASES OF THE CHEST

The Council on Postgraduate Medical Education of the American College of Chest Physicians will present the following Postgraduate Courses on Diseases of the Chest during the period January - April, 1957:

Vanderbilt University, Nashville, Tennessee
January 14 - 18
Mark Hopkins Hotel, San Francisco, California
February 25 - March 1
Bellevue-Stratford Hotel, Philadelphia, Pennsylvania
April 1 - 5

Tuition for each course is $75. The most recent advances in the diagnosis and treatment of chest diseases — medical and surgical — will be presented.

Further information may be obtained by writing to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

NEWS ITEM

Dr. Benjamin O. Morrison attended the Annual Convention of the American College of Gastroenterology, at The Roosevelt in New York City, October 15-20, 1956.

ASPIRIN'S ANTI-INFLAMMATORY ACTION STRESSED IN RHEUMATOID ARTHRITIS

The anti-inflammatory activity of aspirin warrants its continued use in large dosages in the treatment of rheumatoid arthritis, according to an editorial in **Southern Medical Journal** (49: 304, 1956).

Aspirin's well-known analgesic and antipyretic effects supplement its anti-inflammatory action in symptomatic management of the arthritic patient, the journal states. In large doses, however, its primary activity is anti-inflammatory, which also accounts for its major therapeutic usefulness in treating rheumatic fever. Gastric symptoms "are rarely of sufficient severity to require a stoppage of the treatment."

Results of the two-year study by the Joint Committee of the Medical Research Council and Nuffield Foundation (Great Britain) are cited as evidence of aspirin's therapeutic effectiveness in early cases of rheumatoid arthritis. A comparison of two series of patients, one treated with cortisone and the other with aspirin, revealed "remarkedly little to choose between cortisone and aspirin in the management of this group of patients." Evaluations were made in terms of joint tenderness, strength of grip, tests of dexterity and clinical assessments of the disease's activity.

Aspirin seems to have an ACTH-like effect, the editorial says. It points to a number of experiments showing that aspirin stimulates the pituitary gland to put out adrenocorticotropic hormones, thereby stimulating adrenal cortical activity.

CHILDREN UNDER FIVE RESPOND WELL TO POLIO VACCINE

Polio Vaccine appears to work as well in infants and preschool children as it does in school children, providing they receive at least two and preferably three inoculations, a recent Michigan study has shown.

Field trials of the Salk polio vaccine were carried out in 1954 among first, second and third grade children. Although the age range was extended greatly in 1955, little information has been obtained about responses in different age groups.

Since most immunization programs are begun in the early months of life, information about the response of infants to the polio vaccine is important, Gordon C. Brown, Sc.D., and Donald C. Smith, M.D., Ann Arbor, Mich., said.

They found that three inoculations evoked a satisfactory response in infants and in children one to five years old. Two inoculations appeared to be less effective and one injection in infants was definitely inadequate, they said in the June 2 Journal of the American Medical Association.

A booster inoculation six months after the first series raised the number of antibodies in the children's blood regardless of how many initial inoculations they had, or how they responded to the first shots. Even those who did not respond to the original vaccination did develop antibodies after the booster shot.

INCREASE IN SCURVY CAUSED BY BABY FEEDING CHANGES

The abandonment of "pot liquor" and "milk from the family cow" as traditional baby foods — and neglect of daily orange juice — were blamed for an increase in scurvy among infants in Tennessee.

Dr. Calvin Woodruff said in the June 2 Journal of the American Medical Association that the number of cases seen at Vanderbilt University Hospital, Nashville, had jumped from eight in 1926-34 to 45 in 1950-54. From 1926 to 1954 there was a total of 103 cases.

Scurvy, caused by a deficiency of ascorbic acid, occurs most frequently between the ages of seven and 11 months. It can produce bone deformities, and its symptoms sometimes are like those of poliomyelitis.

Ascorbic acid can be obtained from several sources, including orange juice (probably the best source), fresh unpasteurized milk, tomato juice, and even "pot liquor," the water in which green vegetables and fat meat have been boiled.

The abandonment of these old feeding customs and the sterilization of milk—without substituting anything for these sources of ascorbic acid—have resulted in the increase of scurvy.

MAGNESIUM SALTS MAY OVERCOME DELIRIUM TREMENS

Magnesium salts—used to prevent "grass staggers" in cattle after early spring grazing—may also help overcome delirium tremens in chronic alcoholism, according to a report by the American Medical Association's council on foods and nutrition.

The normal adult human body contains less than an ounce of magnesium, but a deficiency can produce muscle twitching, excessive nervousness, tremor, delirium, and even convulsions, Dr. Edmund B. Flink, Minneapolis, said in the report of the council in the April 21 Journal of the American Medical Association.

The human symptoms are similar to those produced in cows, calves, and horses suffering from "grass staggers," a magnesium deficiency illness usually occurring one to two weeks after the livestock begins grazing on new spring grass. Symptoms of "grass staggers" include nervousness, restlessness, grazing away from the herd, lack of appetite, muscle twitching, unsteady gait, spreading of the hind limbs, gnashing of teeth, a wild look, and constant lowing.

Chronic alcoholism appears to be an important cause of magnesium deficiency symptoms in humans, Dr. Flink said. The symptoms can also result from prolonged intravenous administration of magnesium-free fluids.

6,000 ALIEN PHYSICIANS TRAINING IN U. S.

More than 6,000 foreign physicians are in the United States this year to complete their professional training as interns or residents, while more than 1,700 Americans are studying at foreign medical schools.

A survey of foreign physicians in the U. S. was made by the Institute of International Education and the American Medical Association, and was reported in the May 26 Journal of the A.M.A. by Dr. James E. McCormack and Arthur Feraru, New York.

The study showed that 1,730 Americans are studying in 26 different countries. Switzerland, Italy, Canada, Netherlands and Belgium each have more than 100 American students. The University of Bologna (Italy) has the largest number, 243 American medical students. The next five are the University of Geneva (Switzerland), 215; University of Lausanne, (Switzerland, 159; McGill University (Canada), 159; University of Leiden (Netherlands), 70, and University of Heidelberg (Germany), 54.

The 6,033 foreign physicians in the U. S. are training in 764 hospitals listed as approved for internships and residencies by the A.M.A.'s council on medical education and hospitals. This is almost a thousand more physicians than the 5,035 reported in 1954-55. Approximately 47 per cent first came here this year.

PILLS MAY SOME DAY REPLACE INSULIN INJECTIONS

A sulfonamide derivative which can be taken in pill form may some day replace insulin injections for certain types of diabetics, two Indianapolis physicians have reported.

Experiments with the drug, carbutamide, have been widespread in Germany and are now being carried on among several hundred diabetics in this country. Its exact role in the treatment of diabetes is still to be determined, but it appears to be helpful in some cases of the disease, the doctors said in the April 14 Journal of the American Medical Association.

On the basis of research done so far, it is unlikely that the drug will be on sale for some time. At present it is available only for experimental purposes. Many other products have been tested in the hope of replacing insulin injections but none have worked well enough.

Carbutamide (called BZ-55 by its German investigators) and a related compound, tolbutamide, which is also being tested, are classed with the germ-killing sulfonamide derivatives which have been shown to lower blood sugar levels. This lowering—essential to life for the diabetic—is now done through diet and by giving insulin injections to make up the shortage of insulin resulting from either poor production by the pancreas or destruction of insulin by some unknown agent in the body.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

WOMAN'S AUXILIARY TO THE SOUTHERN MEDICAL ASSOCIATION

RESPONSE TO THE WELCOME ADDRESS

By Mrs. Edwin R. Guidry

From the birthplace of the Woman's Auxiliary to the Southern Medical Association I bring you greetings upon the occasion of this golden anniversary of Southern and a "We're delighted to be here" response in the name of all the visiting auxiliaries to the welcome just accorded us.

I also bring you greetings from Mrs. Arthur A. Herold, our first lady of the Auxiliaries in

Louisiana and one of your past-presidents, who wants you to know that only her doctor's sternest orders prevent her from being with you on this occasion. Mrs. Wiley A. Buffington, another former Southern Auxiliary president, also wishes to be remembered to you and to extend her best wishes for a successful convention. It is a matter of great personal regret that our Alma, who so recently guided the Woman's Auxiliary to the Southern Medical Association through a successful year, is not able to be with us especially at the presentation of the George D. Feldner trophy later on today. From her too, I bring warmest greetings and best wishes for success.

You can see how humble I feel coming from such a distinguished line of active Southern Auxiliarites. When I attended my first Southern convention, which incidentelly was my very first auxiliary work after having had the honor of marrying a doctor, I remarked to a friend, "But they are all such important people!" Now after nearly twenty-five years of auxiliary work and a maturer viewpoint, I have come to realize how much I have to learn from such devoted auxiliary members and what a privilege it is to participate in your meetings.

We are all very fortunate in having the committee prepare such wonderful entertainment for us and I thrill to having my store of patriotism replenished with visits to the great buildings of state and to stand upon the hallowed ground of Mount Vernon.

I have taken the opportunity of swelling the Louisiana delegation with our three daughters and am so grateful to you for holding your convention in our nation's capital so that they too could share the privilege of visiting the "greats" of our beloved America.

And, in the not too distant future I look forward to welcoming you all to New Orleans.

Submitted by:
Mildred G. Carter, Chairman
Press and Publicity

The Orleans Club on St. Charles Avenue was the setting Wednesday, November 14, of the meeting and tea for members of the Woman's Auxiliary to the Orleans Parish Medical Society.

Fr. Edward Murphy, S.S.J., author of the "Scarlet Lily", was the guest speaker.

Guests of honor were the Hospital Auxiliary Presidents: Mms. Wiley R. Buffington, Eye, Ear, Nose and Throat Hospital; Lawrence D. Kavanagh, Hotel Dieu; Jules Davidson, Sara Mayo Hospital; Francis X. LeTard, Hospitality Club of Ochsner Medical Foundation; H. W. Lengsfield, Touro Infirmary; Walter P. Richardson, DePaul Hospital; Vincent Randazzo, Mercy Hospital, and Colles Stowell, Crippled Children's Hospital.

Hostesses for the afternoon were Mms. Dan W. Beacham, Walter F. Becker, Philips J. Carter, Julius T. Davis, Jr., Andrew V. Friedrichs, Abram O. Goldsmith, Frederick Fenno, William H. Harris, Sr., William H. Harris, Jr., George F. Sustendal, James T. Swindle, Charles R. Robinson, Norton W. Voorhies, Marshall Michel, and George Feldner.

The Auxiliary's next meeting will be held Wednesday, December 12.

BOOK REVIEWS

Atlas of Roentgen Anatomy of the Skull; by Lewis E. Etter, M. D., with a Section on The Radiographic Anatomy of the Temporal Bone by J. Brown Farrior, M. D. and a Section on The Roentgen Anatomy of the Skull in the Newborn Infant by Samuel G. Henderson, M. D. and Louise S. Sherman, M. D., Springfield, Ill., Charles C Thomas, 1955, Pp. 215, illus., Price $12.50.

The primary purpose of this atlas is to clearly illustrate the detailed anatomy of the various component overlapping shadows seen in radiographs of the skull and face. To accomplish this the various bones of the skull were disarticulated and the individual bones were photographed and radiographed in the standard positions commonly used in making roentgenograms of the skull and face. The dry skull with the individual bone removed was then photographed and radiographed in a similar manner. After replacing the individual bone the same procedure was repeated.

The book contains seven sections. Section I contains general views of the skull. Section II shows general views of the skull with analysis of individual bone components. Section III deals with the paranasal sinuses and mastoids.

Section IV by Dr. Farrior is concerned with the radiographic anatomy of the temporal bone and petrous mastoid portion. Dr. Etter's plan of illustrating anatomy is followed here. Several beautiful colored illustrations are included in this section. The various component portions of the temporal bone have been separated for more detailed study.

Section V explores the orbits and optic foramina. In Section VI the maxillofacial components of the skull are discussed.

Section VII, by Drs. Henderson and Sherman illustrates the roentgen anatomy of the skull in the newborn infant. Because of the difficulty of obtaining roentgenograms on infants, these reproductions do not show the excellent detailed structure noted in the remainder of the book. The table covering the embryology of the skull is an important addition to this section.

In general the reproductions of photographs and radiographs are excellent. The detail shown is unusual and outstanding. The text is clear and

concise. A bibliography and an adequate index are included. This atlas should prove extremely valuable to all interested in this subject and a necessity to the radiologist and to the surgeon who operates on the skull and face.

J. N. ANÉ, M. D.

Stellate Ganglion Block; by Daniel C. Moore, M. D., Springfield, Illinois, Charles C Thomas, 1954, Pp. 280, Price $10.50.

This book has detailed descriptions and illustrations depicting various techniques of stellate ganglion block as well as a summarization of the complications and their treatment. It adequately documents the clinical indications and uses. The anatomical illustrations are excellent. It includes a short discussion of the physiology of the cervicothoracic portion of the sympathetic system. It is a very useful and handy reference for persons interested in the diagnostic and therapeutic utilization of stellate blocks.

CHARLES C. ABBOTT, M. D.

A Textbook of Medicine; Editors: Russell L. Cecil, M. D., and Robert F. Loeb, M. D., Ninth Ed., Illus., W. B. Saunders Company, Philadelphia, Penna., 1955, Pp. 1,786, Price $15.00.

This book remains outstanding as a textbook for medical students as well as postgraduate students of medicine. The ninth edition has been considerably improved by the addition of some of the recently delineated virus infections. The section on xanthomatosis is welcome and well done.

A mature opinion is given on the value of the various antibiotics in specific infectious disease. Such opinion was unavailable for the eighth edition.

The only deficiencies that this reviewer could find in the book were found in the section on diseases of the reticulo-endothelial system. The section on diseases of the spleen is quite confusing and the medical student would probably benefit by not reading this section. There is no discussion in the book of granuloma inguinale.

The book is most valuable as a frame of reference and as such is invaluable in the study of medicine.

FRED M. HUNTER, M. D.

PUBLICATIONS RECEIVED

Grune & Stratton, N. Y.: The Person Behind the Disease, by Julius Bauer, M. D.

Lange Medical Publications, Los Altos, Calif.: Principles of Clinical Electrocardiography, by Mervin J. Goldman, M. D.

The C. V. Mosby Co., St. Louis: The Management of Fractures, Dislocations and Sprains, by John Albert Key, M. D., and H. Earle Conwell, M. D. (6th edit.); Practical Pediatric Dermatology, by Morris Leider, M. D.; Handbook of Pediatric Medical Emergencies, by Adolph G. DeSanctis, M. D., with the collaboration of Charles Varga, M. D., and ten contributors, (2nd edit.).

W. B. Saunders Co., Phila.: Clinical Examinations in Neurology, by members of the Sections of Neurology and Section of Physiology, Mayo Clinic and Mayo Foundation for Medical Education and Research.

Charles C Thomas, Publisher, Springfield, Ill.: Chemosurgery in Cancer, Gangrene and Infections, by Frederic E. Mohs, M. D.; Wire Brush Surgery in the Treatment of Certain Cosmetic Defects and Diseases of the Skin, by James W. Burks, Jr., M. S., M. D.

INDEX TO VOLUME ONE HUNDRED AND EIGHT

January, 1956—December, 1956

A

ACARAPHOBIA
dermal parasitosis, delusions of, manifested by folie à deux, (McAndrews & others) 279
ACETAZOLAMIDE
treatment of epilepsy, 304-E
ACETYLSALICYLIC ACID
toxicity, allergic reaction, (Cazort) 218
ACID
p-aminosalicylic, and streptomycin, (PAS) treatment of pulmonary tuberculosis, (Monte & others) 289
ACIDOSIS
in diabetes, prevention and treatment, (Rippy) 185
ACTH: See Corticotropin
ADRENALS
cortex, clinical relationship to diabetes mellitus, (Gordon) 405
AFIBRINOGENEMIA: See Blood
ALCOHOLISM
some aspects of, (Ritter) 418
ALLERGY
reactions to common drugs, (Cazort) 215
AMEBIASIS
therapy, achievements in, (McHardy & others) 49
therapy with Win 5047 in patients infected concomitantly with E. histolytica and Strongyloides stercoralis, (McHardy & others) 27
ANESTHESIA
local, transvaginal pudenal nerve block, an improved approach in obstetrics and gynecology, (Wilds) 204
ANEURYSM
aortic, thoracic, popliteal, replacement with graft, (Kahle) 351
ANGINA PECTORIS
radioactive iodine for intractable, (Goldman) 337
ANOMALIES: See also Abnormalities under names of specific organs
coarctation of the aorta, congenital, surgical correction, report of 5 cases, (Beskin) 400
ANTIBIOTICS: See also Penicillin, Streptomycin
therapy in amebiasis, (McHardy & others) 49
AORTA
coarctation, surgery, (Beskin) 79
coarctation, congenital, surgical correction; report of 5 cases, (Beskin) 400
APPARATUS
artificial heart-lung, an approach to open heart surgery; preliminary report, (Hyman & others) 134
APPENDICITIS
in newborn infant, (Harris) 446
APPENDIX
disorders of, some unusual cases, (Harris) 446
APRESOLINE: See Hydralazine
ARMED FORCES
dependents, effects of Dependents' Medical Care Act on physicians, 424-E
ARTERIES
coronary disease, unexpected sudden death in, (Matthews & LeDoux) 10
grafts, bank for collection and storage in New Orleans, (Kahle) 271
grafts, the artery bank and arterial grafts. II Arterial grafting, (Kahle) 352
replacement, Ivalon femoral artery prosthesis, (Foster & others) 214
ARTERY BANK: See Arteries, grafts
ASPIRIN: See Acetylsalicylic Acid
AUTOPSIES
analysis of 2229, circulatory system, cardiac, pulmonary, renal, and related deaths at Touro Infirmary, January 1, 1945, through August 31, 1955, (Lubritz) 157

B

BARBITURATES
derivatives, for epilepsy, 304-E
BENEMID: See Probenecid
BILE DUCT
calculi, indications for exploration at cholecystectomy, (Stander & Cacioppo) 94
calculi, surgery of biliary tract, (Mahorner) 75
BILIARY SYSTEM
malabsorption syndrome, (Kleckner) 359
surgery, (Mahorner) 75
BIOPSIES
needle, use and misuse in malignant disease, (Krementz) 293
BITES
black widow spider, hemiplegia from, (Davis) 123
BLADDER
rupture, in infant in the neonatal period; report of case, (Kroll & others) 58
BLEEDING: See Hemorrhage
BLOOD
afibrinogenemia, acquired, with Couvelaire uterus, following cortisone therapy and pitocin induction for an Rh isosensitization, (McHenry) 169
BLOOD—Continued
coagulation mechanism and defects in abnormal bleeding, (Hayes) 367
BLOOD PRESSURE, HIGH
treatment, out-patient, (Moyer) 231
BLOOD TRANSFUSION
risks of, 264-E
BODY
bequest at death to Louisiana State Anatomical Board, (Cummins) 171
BOWELS: See Intestines
BURSITIS
cause of painful shoulder, (Newport & others) 20

BOOK REVIEWS

Albritton, E. C., (Editor), Standard Values in Nutrition and Metabolism, 74
Alexander, Harry L., Reactions with Drug Therapy, 112
Animals
Animal Agents and Vectors of Human Disease, (Faust) 183
Arteries
Angiographic Localization of Intracranial Masses, (Ecker & Riemenschneider) 310
Atlas
Atlas of Roentgen Anatomy of the Skull, (Etter) 469
Human Brain in Sagittal Section, (Singer & Yakovlev) 34
Auxiliary Heart, (Wasson) 112
Aviation
Doctors in the Sky; The Story of the Aero Medical Association, (Benford) 148
Basic Surgical Skills. A Manual with Appropriate Exercises, (Tauber) 432
Benford, Robert J., with foreword by John F. Fulton, Doctors in the Sky; the Story of the Aero Medical Association, 148
Blood Vessels
Auxiliary Heart, (Wasson) 112
Brain
Angiographic Localization of Intracranial Masses, (Ecker & Riemenschneider) 310
Human Brain in Sagittal Section, (Singer & Yakovlev) 34
Calkins, Leroy A., Normal Labor, 394
Cancer
Differential Diagnosis of Leukoplakia, Leukokeratosis, and Cancer in the Mouth, (Welsh) 230
Canizares, Orlando, Modern Diagnosis and Treatment of the Minor Venereal Diseases, 74
Cardiovascular System: See Blood Vessels; Heart
Cecil, Russell L., and Loeb, R. F., Textbookof Medicine, 470
Cerebral Vascular Diseases; Transactions of Conference Held under the Auspices of the American Heart Association, (Lucky, Editor) 183
Clinical Significance of Disturbances in the Delivery of Sweat, (Sulzberger & Herrmann) 34
Communicable Diseases
Pomp and Pestilence. Infectious Disease, Its Origins and Conquest, (Hare) 182
Conant, N. F., and others, Manual of Clinical Mycology, 230
Counseling in Medical Genetics, (Reed) 230
Dermatology
Psychocutaneous Medicine, (Obermayer) 183
Reactions with Drug Therapy, (Alexander) 112
Therapy of Skin Tuberculosis, (Riehl & Kopf) 184
Diagnosis
Differential Diagnosis of Leukoplakia, Leukokeratosis, and Cancer in the Mouth, (Welsh) 230
Differential Diagnosis, the Interpretation of Clinical Evidence, (Harvey & Bordley) 270
Modern Diagnosis and Treatment of the Minor Venereal Diseases, (Canizares) 74
Disease: See also Diagnosis
Animal Agents and Vectors of Human Disease, (Faust) 183
Cerebral Vascular Diseases; Transaction of Conference Held under the Auspices of the American Heart Association, (Lucky, Editor) 183
Pomp and Pestilence. Infectious Disease, Its Origin and Conquest, (Hare) 182
Potassium Metabolism in Health and Disease, (Holley & Carlson) 148
Doctors: See Physicians
Drugs
Reactions with Drug Therapy, (Alexander) 112
Ecker, Arthur, and Riemenschneider, Paul A., Angiographic Localization of Intracranial Masses, 310
Etter, Lewis E., Atlas of Roentgen Anatomy of the Skull, 469
Faust, Ernest Carroll, Animal Agents and Vectors of Human Disease, 183
Fish, John S., Hemorrhage of Late Pregnancy, 182
Food: See Nutrition
Genetics
Counseling in Medical Genetics, (Reed) 230
Gray, Laman A., Vaginal Hysterectomy, Indications, Technique, and Complications, 350
Hare, Ronald, Pomp and Pestilence. Infectious Disease, Its Origins and Conquest, 182
Harvey A. McGehee, and Bordley, James, Differential Diagnosis, the Interpretation of Clinical Evidence, 270

Health
  Potassium Metabolism in Health and Disease, (Holley & Carlson) 148
Heart
  Auxiliary Heart, (Wasson) 112
Hemorrhage of Late Pregnancy, (Fish) 182
Holley, Howard L., and Carlson, W. E., Potassium Metabolism in Health and Disease, 148
Human Brain in Sagittal Section (Singer & Yakovlev) 34
Hysterectomy
  Vaginal Hysterectomy, Indications, Technique, and Complications, (Gray) 350
Infectious Diseases: See Communicable Disease
Ion Exchange and Adsorption Agents in Medicine. The Concept of Intestinal Bionomics, (Martin) 270
Kidneys
  Reflections on Renal Function, (Robinson) 184
Labor: See Obstetrics
Leukokeratosis
  Differential Diagnosis of Leukoplakia, Leukokeratosis, and Cancer in the Mouth, (Welsh) 230
Leukoplakia
  Differential Diagnosis of Leukoplakia, Leukokeratosis, and Cancer in the Mouth, (Welsh) 230
Manual
  Basic Surgical Skills. A Manual with Appropriate Exercises, (Tauber) 432
  Manual of Clinical Mycology, (Conant & others) 230
Martin, Gustav J., Ion Exchange and Adsorption Agents in Medicine. The Concept of Intestinal Bionomics, 270
Masserman, Jules H., The Practice of Dynamic Psychiatry, 350
Mechanism of Labor, (Rydberg) 111
Medicine
  Psychocutaneous Medicine, (Obermayer) 183
  Textbook of Medicine, (Cecil & Loeb) 470
Metabolism
  Potassium Metabolism in Health and Disease, (Holley & Carlson) 148
  Standard Values in Nutrition and Metabolism, (Albritton) 74
Modern Diagnosis and Treatment of the Minor Venereal Diseases, (Canizares) 74
Moore, Dani. C., Stellate Ganglion Block, 470
Mouth
  Differential Diagnosis of Leukoplakia, Leukokeratosis, and Cancer in the Mouth, (Welsh) 230
Mycology
  Manual of Clinical Mycology, (Conant & others) 230
Normal Labor, (Calkins) 394
Nutrition
  Standard Values in Nutrition and Metabolism, (Albritton) 74
Obermayer, Maximillian E., Psychocutaneous Medicine, 183
Obstetrics
  Mechanism of Labour, (Rydberg) 111
  Normal Labor, (Calkins) 394
Parasitology
  Animal Agents and Vectors of Human Disease, (Faust) 183
Physicians
  Doctors in the Sky; The Story of the Aero Medical Association, (Benford) 148
Physiology
  Reflections on Renal Function, (Robinson) 184
Pomp and Pestilence. Infectious Disease, Its Origins and Conquest, (Hare) 182
Potassium Metabolism in Health and Disease, (Holley & Carlson) 148
Practice of Dynamic Psychiatry, (Masserman) 350
Pregnancy
  Hemorrhage of Late Pregnancy, (Fish) 182
Psychiatry
  Practice of Dynamic Psychiatry, (Masserman) 350
  Psychocutaneous Medicine, (Obermayer) 183
  Twentieth Century Psychiatry, (White) 230
Reactions with Drug Therapy, (Alexander) 112
Reed, Sheldon C., Counseling in Medical Genetics, 230
Reflections on Renal Function, (Robinson) 184
Riehl, Gustav, and Kopf, Oswald: The Therapy of Skin Tuberculosis, 184
Robinson, James R., Reflections on Renal Function, 184
Roentgenology
  Angiographic Localization of Intracranial Masses, (Ecker & Riemenschneider) 310
  Atlas of Roentgen Anatomy of the Skull, (Etter) 469
Rydberg, Erik, The Mechanism of Labor, 111
Singer, Marcus, and Yakovlev, Paul I., The Human Brain in Sagittal Section, 34
Skin: See Dermatology
Standard Values in Nutrition and Metabolism, (Albritton) 74
Stellate Ganglion Block, (Moore) 470
Sulzberger, Marion B., and Herrmann, Franz, Clinical Significance of Disturbances in the Delivery of Sweat, 34
Surgery
  Basic Surgical Skills. A Manual with Appropriate Exercises, (Tauber) 432
Sweat
  Clinical Significance of Disturbances in the Delivery of Sweat, (Sulzberger & Herrmann) 34
Textbook of Medicine, (Cecil & Loeb) 470
Therapeutics
  Modern Diagnosis and Treatment of the Minor Venereal Diseases, (Canizares) 74
  Reactions with Drug Therapy, (Alexander) 112
  Therapy of Skin Tuberculosis, (Riehl & Kopf) 184
Tuberculosis
  Therapy of Skin Tuberculosis, (Riehl & Kopf) 184
Tumors
  Angiographic Localization of Intracranial Masses, (Ecker & Riemenschneider) 310
Twentieth Century Psychiatry, (White) 230
Vaginal Hysterectomy, Indications, Technique, and Complications, (Gray) 350
Venereal Disease
  Modern Diagnosis and Treatment of the Minor Venereal Diseases, (Canizares) 74
Wasson, William Walter, The Auxiliary Heart, 112
Welsh, Ashton L., Differential Diagnosis of Leukoplakia, Leukokeratosis, and Cancer in the Mouth, 230
White, William A., Twentieth Century Psychiatry, 230
Wright, Irving S., Chairman; Lucky, E. Hugh, Editor, Cerebral Vascular Diseases; Transactions of Conference Held under the Auspices of the American Heart Association, 183

C

CALCULI: See Gallbladder
CANCER: See also under name of organ or region affected
  biliary tract, surgery, (Mahorner) 75
  diagnosis, use and misuse of needle biopsy, (Krementz) 293
  of appendix and cecum, primary, (Harris) 446
  pancreas, surgical aspects, (Child & Donovan) 195
  stomach, comparison of malignant, benign, and medically managed groups of gastric ulcer at Vanderbilt University Hospital, (Sawyers & others) 433
CARBUTAMIDE
  treatment of diabetes, 388-E
CARCINOMA: See Cancer
CARDIAC: See Heart
CARDIOVASCULAR SYSTEM
  analysis of 2229 autopsies with special emphasis on circulatory system and allied deaths, (Lubritz) 157
CATHETERIZATION
  cardiac, diagnosis of atrial septal defect, (Hyman) 85
  cardiac, value of, in determining need for valvulotomy in mitral stenosis, (Hyman) 190
CESAREAN SECTION
  review of 1000 at Southern Baptist Hospital, New Orleans, (Davis & Ward) 286
CHARITY HOSPITAL OF LOUISIANA
  abdominal wound complications in obstetric and gynecologic surgery, January 1946 through December 1955, (Nix & others) 395
  experiences with endometriosis 1939-1953, (McCall & Dolan) 43
  report of committee, East Baton Rouge Medical Society on need for charity hospital facilities, (Peek & others) 60
  review of cases with shigellosis admitted to the pediatric services, during 1955, (Curry & Sappenfield) 442
CHILDREN: See also Infants; Pediatrics; under names of specific diseases
  cardiac handicapped, problem of in Louisiana, (Berenson) 173
  cross-eyed, development of vision, (Ellis & Haik) 450
  enuresis and pyuria, (Beckman) 90
CHOLE: See Bile Ducts; Biliary System; Gallbladder
CHOLECYSTITIS: See Gallbladder
CHOLEDOCHOLITHIASIS: See Bile duct
CHOLELITHIASIS: See Gallbladder, calculi
COAGULATION: See Blood, coagulation
COLCHICINE
  treatment of gout, (Talbott) 149
CONGENITAL, defects: See Anomalies, congenital; also under organ, region, or structure affected
CORONARY: See Arteries, coronary
CORTICOTROPIN (ACTH)
  effects of, and cortisone in carbohydrate metabolism, and production of diabetes, (Gordon) 405
CORTISONE
  effects of, and ACTH in carbohydrate metabolism and production of diabetes, (Gordon) 405
  therapy, and pitocin induction for Rh incompatibility, with acquired afibrinogenemia and Couvelaire uterus, (McHenry) 169
CROSSED EYES: See Strabismus
CUSHING'S SYNDROME
  carbohydrate metabolism, relationship of the adrenal cortex to diabetes mellitus, (Gordon) 405
CYANOSIS
  pathogenesis, observations on atrial septal defect, (Hyman) 85

D

DEATH
  bequest of the body at, to Louisiana State Anatomical Board, (Cummins) 171
  unexpected, sudden, and acute myocardial infarction and acute myocardial ischemia, (Matthews & LeDoux) 10
DELIVERY: See Labor
DELUSIONS
  of dermal parasitosis (acarophobia), manifested by folie à deux, (McAndrews & others) 279
DENTISTRY
  care of teeth in cleft palate cases, (Laguaite) 119
DERMATITIS
  contact, drug reaction, (Cazort) 216
  Industrial: See Industrial Dermatoses

DIABETES MELLITUS
acidosis, prevention and treatment of ketosis, (Rippy) 185
clinical relationship of adrenal cortex to, (Gordon) 405
complications, long range, of poorly controlled diabetes, (Murison) 137
status of antidiabetic sulfonamide compounds, 388-E
DIAGNOSIS
of gout, and treatment, (Talbott) 149
DISABILITY
determinations under HR 7225, threat to American medicine, 29-E
total after age 50, provision of recently enacted Social Security amendments law, dangerous feature, 344-E
DISEASE: See also under names of specific diseases
pancreas, surgical aspects, (Child & Donovan) 195
DIVERTICULA
of appendix, (Harris) 446
DOCTORS: See Physicians
DRUGS: See also under names of specific drugs
allergic reactions, (Cazort) 215
antithyroid, propylthiouracil and methimazole (tapazol) in "therapeutic trial" as a criterion in hyperthyroidism, (Singer) 384
hypotensive, review of pharmacodynamics of antihypertensive therapy, (Moyer) 231
new—when should we use them, (Langford) 301
DYSTOCIA: See Labor

E

EAST BATON ROUGE PARISH
charity hospital committee report, (Peek & others) 60
ELECTROCARDIOGRAM: See Heart
EMBOLISM
pulmonary, incidence in 2229 autopsies, (Lubritz) 157
ENDOMETRIOSIS
pelvic, review of private cases and at Charity Hospital of Louisiana, New Orleans, 1939-1953, (McCall & Dolan) 43
ENZYMES
serum transaminase as an aid in diagnosis of myocardial infarction, (McHardy & Stotler) 440
EPILEPSY
atypical and minor seizure, (Moossy) 331
treatment, status of drugs, 304-E
EYES
cross-eyed child, development of vision, (Ellis & Haik) 450
disease, management of the red eye in general practice, (Newell) 211
hyphema, traumatic, (Rosenthal) 99
hyphema, traumatic, (Wilkinson) 56

F

FECALITH
in rectosigmoid pouch, causing dystocia, (Pallés) 262
FEDERAL
Civil Defense Administration Medical Services, (Head) 245
Legislation: See Laws and Legislation, federal
FEMUR
fractures of femoral neck, head prosthesis in, (Rose) 276
FEVER
etiology, need for urologic survey, (Beckmann) 90
FOOD
malabsorption syndrome, (Kleckner) 359
FOLIE A DEUX: See Insanity, communicated
FRACTURES
femoral neck, head prosthesis in, (Rose) 276
hip, reconstruction procedures, (Banks) 415

G

GALLBLADDER
calculi, surgery, indications for, (Mahorner) 75
surgery, a comparative study, (Stander & Cacioppo) 94
GALLSTONES: See Gallbladder, calculi
GANGLION (nerve)
block, use of agents in hypertension, plus rauwolfia, (Moyer) 237
GASTRIC: See Stomach
Ulcer: See Peptic Ulcer
GENERAL PRACTITIONER: See Physician, practicing
GERIATRICS: See Old Age
GOUT
diagnosis and treatment, (Talbott) 149
GOVERNMENT: See United States
GRAND MAL: See Epilepsy
GYNECOLOGY
surgery, abdominal wound complications in, (Nix & others) 395
surgery, grading of indication and choice of procedure, at a private hospital, (Dougherty & others) 208
transvaginal pudendal nerve block, an improved approach, (Wilds) 204

H

HEART
anomalies, tetralogy of Fallot, surgery, (Beakin) 79
artificial heart-lung, plastic, an approach to open heart surgery, (Hyman & others) 134
cardiac handicapped children in Louisiana, problem of, (Berenson) 173
catheterization, atrial septal defect, observations on 41 patients diagnosed, (Hyman) 85
catheterization, value in determining need for valvulotomy in mitral stenosis, (Hyman) 190
disease and allied causes of death, analysis of cases autopsied, (Lubritz) 157
HEART—Continued
Disease (Coronary): See Arteries, coronary
electrocardiogram, new explanation of the absence of P-waves in a case of Tawara-node rhythm with retrograde conduction, (Peters) 156
Infarction: See Myocardium
Mechanical: See Heart, artificial
surgery, in a community hospital, (Beakin) 79
Valves: See Mitral Valve
HEMATURIA
etiology and treatment, (Hatcher) 328
need for accurate urologic study in presence of, (Beckmann) 90
HEMIPLEGIA
from bite of black widow spider, (Davis) 123
HEMORRHAGE: See also under names of diseases, regions, and organs affected
mechanism and differential diagnosis of abnormal bleeding, (Hayes) 367
HERNIA
surgery, principles in repair, (D'Ingianni) 381
HIP
prosthesis, head, in fractures of femoral neck, (Rose) 276
reconstruction procedures, (Banks) 415
HOSPITALS
charity, Louisiana has highest number of charity beds per capita of any state, 142-E
charity, report of committee East Baton Rouge Medical Society as to need for facilities in East Baton Rouge Parish, (Peek & others) 60
HYDANTOIN
derivatives in treatment of epilepsy, 304-E
HYDRALAZINE HYDROCHLORIDE
treatment of hypertension with rauwolfia and apresoline, (Moyer) 235
1-HYDRAZINOPHTHALAZINE: See Hydralazine
HYPERTENSION: See Blood Pressure, High
HYPERTHYROIDISM
"therapeutic trial" as a diagnostic criterion in, (Singer) 384
HYPHEMA
traumatic, (Rosenthal) 99
traumatic, (Wilkinson) 56
HYPOTHYROIDISM
induced with I^{131} in euthyroid patients with intractable angina pectoris, (Goldman) 337
HYSTERECTOMY: See Uterus, surgery

I

INDUSTRIAL DERMATOSES
dermatitis due to chrome, cement, rubber, chlorinated hydrocarbons, (Henington & others) 320
INFANTS, NEWBORN
appendicitis in, (Harris) 446
rupture of urinary bladder in 5 day old premature, (Kroll & others) 58
INFARCTION: See Myocardium
INJURIES: See Fracture; Trauma; and under organ, region, or structure affected
INSANITY
communicated, folie à deux, delusions of dermal parasitosis, (acarophobia), (McAndrews & others) 279
INSURANCE
compulsory Social Security coverage for physicians versus the Jenkins-Keogh bill, 461-E
INTESTINES
malabsorption syndrome, (Kleckner) 359
obstruction, acute; re-evaluation of therapy and review of 2408 cases at a university hospital, (Tendler & Cartwright) 4
IODINE AND IODINE COMPOUNDS
Lugol's solution, "therapeutic trial" as a diagnostic criterion in hyperthyroidism, (Singer) 384
radioactive, for intractable angina pectoris, (Goldman) 337
IVALON
femoral artery prosthesis, (Foster & others) 214

J

JENKINS-KEOGH BILL
versus compulsory Social Security insurance coverage for physicians, 461-E

K

KETOSIS: See Acidosis
KIDNEYS: See also Urinary System
failure, acute, medical management, (Muslow) 38
function, deterioration in, from hypertension, and improvement with therapy, (Moyer) 231
hydronephrosis, patient with minimal urologic symptoms, (Beckmann) 90

L

LABOR: See also Infants, Newborn; Obstetrics; Pregnancy
dystocia due to fecalith in rectosigmoid pouch, (Pallés) 262
induction, acquired afibrinogenemia with Couvelaire uterus following cortisone therapy and pitocin, (McHenry) 169
LABORATORY
tests in diagnosis in abnormal bleeding, (Hayes) 367
LAWS AND LEGISLATION
Dependents' Medical Care Act, Public Law No. 569, and effect on physicians, 424-E
federal, expansion of government medical care and effects on private practice, (Blasingame) 1
federal, HR 7225, Social Security Amendments of 1955, serious threat to American medicine, 29-E
Jenkins-Keogh Bill versus compulsory Social Security coverage for physicians, 461-E

LITHIASIS: See Calculi (cross reference)
LIVER: See also Bile Ducts
carcinoma of, and surgery, (Mahorner) 75
LOUISIANA
hospitals, highest number of charity beds per capita in U. S., (Peek & others) 60
poliomyelitis situation and vaccination in, 70-E
poliomyelitis vaccination program, (Martin) 35
problem of cardiac handicapped children in, (Berenson) 173
taxation, increase in number levied by State, with highest number of free hospital beds, 142-E
typhoid fever in Richland Parish, 1954-1955 (Orgeron & others) 421
LOUISIANA STATE ANATOMICAL BOARD
form to be executed for bequest of body at death, (Cummins) 171
LOUISIANA STATE MEDICAL SOCIETY
officers for 1955-1956, 144
officers for 1956-1957, 221-E
LUNGS: See also Tuberculosis of Lung
LUPUS ERYTHEMATOSUS
serodiagnosis, reactions with treponemal immobilization test, (Miller) 113
LYMPHOSARCOMA
of appendix, primary, (Harris) 446

M

MALABSORPTION SYNDROME
(Kleckner) 359
MALIGNANCIES: See Cancer; Tumors, malignant
MEDICAL NEWS
33, 73, 110, 146, 179, 227, 269, 307, 348, 392, 428, 466
MEDICARE PROGRAM: See Laws and Legislation
MEDICAL SERVICE
Emergency Medical Service for Civil Defense: See Federal
MEDICINE
practice, big government and some of its effects on private practice, (Blasingame) 1
practice, medical implications of HR 7225, serious threat, 29-E
MENTAL DISORDERS
psychotherapy by the general practitioner, (Stevenson) 129
treatment with reserpine in disturbed geriatric patients, (Villien) 126
treatment, use of promazine in psychiatric practice, (Usdin) 251
METABOLISM
carbohydrate, relationship of the adrenal cortex to diabetes mellitus, (Gordon) 405
I^{131} induced hypometabolism in euthyroid patients with intractable angina pectoris, (Goldman) 337
MITRAL VALVE
stenosis, six commissurotomies in a community hospital, (Beakin) 79
stenosis, value of cardiac catheterization in determining need for valvulotomy, (Hyman) 190
MYOCARDIUM
infarction, acute, and myocardial ischemia, and unexpected sudden death, (Matthews & LeDoux) 10
infarction, cardiac rupture following, 21 cases in 2229 autopsies, (Lubritz) 157
infarction, evaluation of serum transaminase as an aid in diagnosis, (McHardy & Stotler) 440

N

NASAL: See Nose
NEOPLASMS: See Cancer; Tumors; under region or organ affected
NERVES
block, transvaginal pudendal block, improved approach in obstetrics and gynecology, (Wilds) 204
NEUROFIBROMA
of appendix, (Harris) 446
NEWBORN: See Infants, Newborn
NODE
Tawara, rhythm with retrograde conduction, a new explanation of absence of P waves in a case of, (Peters) 156
NOSE
deformities, external nasal and septal deformities, surgical correction, (Anderson & Rubin) 258

O

OBSTETRICS: See also Labor; Pregnancy
surgery, abdominal wound complications in, (Nix & others) 395
transvaginal pudendal nerve block, an improved approach, (Wilds) 204
OBSTRUCTION: See under organ affected
OLD AGE
public assistance in Louisiana; 8 out of every 100 receiving, 142-E
treatment of disturbed geriatric patients with rauwolfia serpentina (reserpine), (Villien) 126
OPERATION: See Surgery; under names of specific organ and disease
ORGANIZATION SECTION
31, 71, 107, 144, 177, 222, 264, 306, 346, 390, 425, 463
ORLON
tube replacement of abdominal aorta in experimental animal, (Kahle) 351
ORTHODONTIA
in cleft palate cases, (Laguaite) 119
OXYTOCIN
induction of labor, elective, acquired afibrinogenemia with Couvelaire uterus following cortisone therapy and pitocin induction for an Rh isosensitization, (McHenry) 169

P

PAIN:
shoulder, (Newport & others) 20
PALATE
cleft, importance of team approach in rehabilitation of, (Laguaite) 119
PANCREAS
disease, surgical aspects of: congenital anomalies, injury by violence, pancreatitis, neoplasms, (Child & Donovan) 195
Inflammation: See Pancreatitis
steatorrhea in malabsorption syndrome, (Kleckner) 359
surgery of the biliary tract, (Mahorner) 75
PANCREATITIS
problem of, surgical aspects, (Schaffarzick) 377
surgical aspects, etiology, management of obstruction to drainage, (Child & Donovan) 195
PARALYSIS
Infantile: See Poliomyelitis
PARASITOSIS
dermal, delusions of, (acaraphobia) manifested by folie à deux, (McAndrews & others) 279
PAS: See Acid, p-aminosalicylic
PEDIATRICS: See also Children; Infants
review of cases of shigellosis admitted to the pediatric service of Charity Hospital at New Orleans during 1955, (Curry & Sappenfield) 442
PELVIS
endometriosis, review of private cases and at Charity Hospital, 1939-1953, (McCall & Dolan) 43
PENICILLIN
toxicity, allergic reaction, (Cazort) 217
PEPTIC ULCER
gastric, problem, comparison of malignant, benign, and medically managed groups at Vanderbilt University Hospital, (Sawyers & others) 433
relationship to pancreatic disease, (Child & Donovan) 195
PETIT MAL: See Epilepsy
PHENACEMIDE
treatment of epilepsy, 304-E
PHENSUXIMIDE
treatment of epilepsy, 304-E
PHENURONE: See Phenacemide
PHOTORADIOGRAPHY: See Thorax, chest x-rays
PHYSICIANS
and the "Medicare" program, Dependents' Medical Care Act, 424-E
compulsory insurance under contemplated Social Security coverage versus Jenkins-Keogh bill, 461-E
practicing, management of the red eye in general practice, (Newell) 211
practicing, use of psychotherapy by general practitioner, (Stevenson) 129
public's opinion of doctors, 105-E
PITOCIN: See Oxytocin
PITUITARY
hypophysectomy, influence upon carbohydrate metabolism and insulin sensitivity, (Gordon) 405
POLIOMYELITIS
vaccination; situation in Louisiana, 70-E
vaccination program in Louisiana, (Martin) 35
PRACTICE OF MEDICINE: See Medicine, practice
PREGNANCY: See also Labor; Obstetrics
vomiting of, 176-E
PROBENECID (benemid)
treatment of gout, (Talbott) 149
PROMAZINE
in psychiatric practice; an early study, (Usdin) 251
PROSTATE
carcinoma, with minimal urologic symptoms, (Beckmann) 90
PROSTHESIS
femoral artery, Ivalon, spiral accordionated tube, (Foster & others) 214
head, in fractures of the femoral neck, (Rose) 276
PROSTHODONTIA
use in cleft palate cases, (Laguaite) 119
PSYCHIATRY
Delusions: See Delusions
promazine, use in psychiatric practice, (Usdin) 251
PSYCHOMOTOR EPILEPSY: See Epilepsy
PSYCHOTHERAPY
by the general practitioner, (Stevenson) 129
PUBLIC OPINION
about doctors, 105-E
PULMONARY: See Lungs
Embolism: See Embolism
Tuberculosis: See Tuberculosis of Lung
PYURIA
in children, need for urologic survey, (Beckmann) 90

R

RADIOACTIVE
Iodine: See Iodine, radioactive
RAUWOLFIA SERPENTINA (alseroxylon: rauwolfia; reserpine)
treatment of hypertension with rauwolfia and in combination with apresoline, or plus ganglionic blocking agents, (Moyer) 234
treatment (reserpine), in disturbed geriatric patients, (Villien) 126

REACTION
false positive: See Syphilis
RECTUM
fecalith in rectosigmoid pouch, causing dystocia, (Palles) 262
RENAL: See Kidneys
RESERPINE: See Rauwolfia serpentina
RETIREMENT
benefits, HR 7225, medical implications serious threat to American medicine, 29-E
Rh FACTOR
incompatibility, cortisone therapy and pitocin induction followed by afibrinogenemia with Couvelaire uterus, (McHenry) 157
RICHLAND PARISH
typhoid fever, 1954-1955, (Orgeron & others) 421
ROENTGEN RAYS
Chest x-rays: See Thorax, chest x-rays
RUPTURE: See also Hernia; under specific organs and regions
bladder, urinary, in the neonatal period; report of case, (Kroll & others) 58

S

SALK VACCINE: See Poliomyelitis
SEPTICEMIA
staphylococcal, two cases with pulmonary involvement; manifestations and treatment, (Leckert & Jastremski) 454
SEPTUM
nasal: See Nose, septum
SERODIAGNOSIS: See Syphilis
SERUM TRANSAMINASE: See Enzymes
SHIGELLOSIS
review of cases admitted to the pediatric services of Charity Hospital at New Orleans during 1955, (Curry & Sappenfield) 442
SHOULDER
painful, common causes, differential diagnosis and treatment, (Newport & others) 20
SOCIAL SECURITY
amendments law, dangerous features in, 344-E
amendments of 1955, medical implications of HR 7225, serious threat to American medicine, 29-E
compulsory insurance coverage for physicians versus the Jenkins-Keogh bill, 461-E
SPEECH
appliances and therapy in rehabilitation of cleft palate cases, (Laguaite) 119
SPIDERS
black widow, hemiplegia from bite, (Davis) 123
SPRUE
malabsorption syndrome, (Kleckner) 359
STAPHYLOCOCCUS
septicemia, with two cases pulmonary involvement; manifestations and treatment, (Leckert & Jastremski) 454
STEROIDS
treatment in malabsorption syndrome, (Kleckner) 359
STOMACH
Ulcer: See Peptic Ulcer
STRABISMUS
development of vision in the cross-eyed child, (Ellis & Haik) 450
STREPTOHYDRAZIDE
treatment of pulmonary tuberculosis, evaluation of, (Monte & others) 289
STRONGYLOIDOSIS
therapy with Win 5047: preliminary report, (McHardy & others) 27
SULFONAMIDES
antidiabetic compounds, status of, 388-E
SURGERY: See also under specific diseases, organs, and operations
abdominal wound complications in obstetric and gynecologic surgery, (Nix & others) 395
biliary tract, (Mahorner) 75
cleft palate, (Laguaite) 119
coarctation of the aorta; report of five cases, (Beskin) 400
gallbladder; a comparative study, (Stander & Cacoippo) 94
gynecologic, grading of indication and choice of procedure for, at a private hospital, (Dougherty & others) 208
hernia, principles in repair, (D'Ingianni) 381
pancreas, problem of pancreatitis; surgical aspects, (Schaffarzick) 377
postoperative complication, pancreatitis, (Child & Donovan) 201
transvaginal pudendal nerve block; an improved approach in obstetric and gynecologic surgery, (Wilds) 204
SYPHILIS
serodiagnosis, recent advances in tests, (Hauser & Godelfer) 311
serodiagnosis, significance at the present time of the diagnosis of biologic false positive reaction, (Miller) 113

T

TAX
in Louisiana, burden out of proportion to what it is in other states, 142-E
TENDONS
tears, rotator cuff and tenosynovitis as cause of painful shoulder, (Newport & others) 20
TETRALOGY OF FALLOT: See Heart, anomalies
THERAPY
amebiasis, achievements in, (McHardy & others) 49
gout, (Talbott) 149
medical management of acute renal failure
strongyloidosis with Win. 5047, (McHardy & others) 27
THORAX
chest x-rays, routine photoradiography; its value to the practicing physician, (Jacobs) 92
THYROID
I^{131} induced hypometabolism in euthyroid patients with intractable angina pectoris, (Goldman) 337
propylthiouracil and tapazol used in "therapeutic trial" as a diagnostic criterion in hyperthyroidism, (Singer) 384
TRANQUILIZING DRUGS: See also under name of specific drugs
promazine in psychiatric practice, (Usdin) 251
TRANSFUSIONS: See Blood Transfusion
TRAUMA: See also Wounds; under name of specific disease and organ, region, or structure affected
eye, hyphema, (Rosenthal) 99
eye, hyphema, (Wilkinson) 56
pancreas, surgical aspects of injury by violence, (Child & Donovan) 195
TREPONEMA
immobilization test, recent advances in serodiagnostic tests for syphilis, (Hauser and Godelfer) 312
immobilization test, significance at the present time of diagnosis of biologic false positive reaction, (Miller) 113
TUBERCULOSIS OF LUNG (pulmonary tuberculosis)
therapy, streptohydrazide, evaluation of, (Monte & others) 289
TUMORS: See also under names of specific organs and types of tumors
Malignant: See also Cancer
malignant, needle biopsy in diagnosis, use and misuse, (Krementz) 293
TYPHOID
in Richland Parish, Louisiana, 1954-1955, (Orgeron & others) 421

U

ULCERS: See Peptic Ulcer
UNITED STATES
big government and some of its effects on private practice, (Blasingame) 1
URIC ACID
increased formation in pathogenesis of gout, (Talbott) 149
URINARY SYSTEM: See also Bladder; Kidneys
urologic symptoms, the patient with minimal, (Beckmann) 90
UROLOGIC SYMPTOMS: See Urinary System
UTERUS
Couvelaire, acquired afibrinogenemia following cortisone therapy and pitocin induction for an Rh isosensitization, (McHenry) 169
surgery, review of 176 hysterectomies at a private hospital, with grading of indication and choice of procedure, (Dougherty & others) 208

V

VALVULOTOMY: See Mitral Valve
VISION
development of, in the cross-eyed child, (Ellis & Haik) 450
VOMITING: See also Pregnancy
of pregnancy, 176-E

W

WOMAN'S AUXILIARY
182, 229, 308, 350, 393, 431, 468
WOUNDS: See also Injuries; and under organs, region, or structure, affected
abdominal, complications, in obstetric and gynecologic surgery, (Nix & others) 395

X

X-RAYS: See Thorax, chest x-rays

AUTHOR INDEX

A

Adams, Jesse E., 433
Alldredge, Rufus H., 20
Anderson, Jack R., 258

B

Banks, T. E., 415
Beckmann, George, 90
Berenson, Gerald S., 173
Beskin, Chas. A., 79,400
Blasingame, F. J. L., 1
Blitz, Oscar, 289
Blum, Jos. E., 289
Braden, Frederick R., 395
Browne, Donovan C., 27, 49

C

Cacioppo, D. V., 94
Campanella, Thomas, 60
Caraway, Chas. T., 421
Cartwright, Robt. S., 4
Cazort, Alan G., 215
Chalstrom, Horace, 58
Child, Chas. G., III, 195
Collins, Conrad G., 395
Cummins, Harold, 171
Curry, Max A., 442

D

Davis, Julius T., 286
Davis, Thos. W., 123
Derbes, Vincent, 279
Dickmnn, F. Norton, Jr., 58
D'Ingianni, Vincente, 381
Dolan, Wilfred G., 43
Donovan, Arthur J., 195
Dougherty, Cary M., 208

E

Ellis, Geo. S., 450

F

Foster, R. P., 214

G

Godelfer, Lucille, 311
Goldman, Allan, 337
Gordon, Douglas, 405

H

Haik, Geo. M., 450
Hardy, H. H., Jr., 214
Harris, W. H., Jr., 446
Hatcher, Henry C., 328
Hauser, Geo. H., 311
Hayes, Daniel W., 367
Head, C. D., Jr., 245
Henington, V. Medd, 320
Hyman, Albert L., 85, 134, 190
Hyman, Edward S., 134

J

Jacobs, Sydney, 92
Jastremski, Bruno, 454
Jung, Rodney, 279

K

Kahle, Reichard, 134, 271, 351
Kennedy, C. Barrett, 320
Kleckner, Martin S., Jr., 359
Krementz, Edward T., 293
Kroll, Vernon R., 58
Kyle, W. W., 208

L

Laguaite, Jeanette K., 119
Langford, Richard B., 301
Leckert, John T., 454
LeDoux, Marion J., 10
Lubritz, Ephraim, 157

Mc

McAndrews, John, 279
McCall, Milton L., 43
McHardy, Gordon, 27, 49
McHardy, Robert J., 27, 49, 440
McHenry, Armand G., Jr., 163
McQuown, Albert L., 60

M

Mahorner, Howard, 75
Martin, J. D., 35, 421
Martine, Rose Mary, 421
Matthews, Edward, 10
Miller, J. Lowry, 113
Monte, Louis A., 289
Moossy, John, 331
Moyer, John H., 231
Murison, Paul J., 137
Muslow, Ike, 38

N

Newell, Frank W., 211
Newport, Jack W., 20
Nix, Frank C., 395

O

Orgeron, J. D., 421

P

Pallés, Louis M., 262
Pearce, M. B., 214
Peek, Gordon, 60
Peters, J. T., 156
Pickell, Frank W., 60

R

Randall, Spears, Jr., 60
Riordan, Daniel C., 20
Rippy, Edwin L., 185
Ritter, Kenneth A., 418
Rose, Robt. M., 276
Rosenberg, Dennis, 134
Rosenthal, J. Wm., 99
Rubin, Wallace, 258

S

Sappenfield, Robt., W., 442
Sawyers, John L., 433
Sayegh, Salem, 134
Schaffarzick, Wm. R., 377
Schudmak, M. A., 208
Scott, H. Wm., Jr., 433
Singer, Irving, 384
Stander, Leonard H., 94
Stevenson, Ian, 129
Stotler, John B., 440
Sustendal, Geo. F., 395

T

Talbott, John H., 149
Tendler, Morton J., 4

U

Usdin, Gene L., 251

V

Villien, L. M., 126

W

Ward, Simon V., 286
Wilds, Preston Lea, 204
Wilkinson, J. A., 56
Williams, O. P., 320